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ASSURANCES AS TO THE ADVANTAGES AND SAFETY OF RADIOACTIVE IODINE TREATMENT OF HYPERTHYROIDISM*

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INTRODUCTION

Although the use of radioactive iodine in the therapy of patients with Graves' disease and in the study of thyroid physiology is now over eight years old the method of treatment has only recently been as widely applied as the results would dictate. The limitation of this form of treatment was largely due to the small availability of isotopes for this purpose until a wide expansion of the field was made possible by supplies from such agencies as the Atomic Energy Commission's installation at Oak Ridge, Tennessee. It is fair to say that no longer need availability or expense deter the extended use of radioactive iodine for specified purposes. Safety in handling the isotopes and expense are largely solved.

Another deterrent to the wider use of the isotope therapy has been the undercurrent of rumors that radioactive iodine "might cause renal damage" ("radiation nephrosis"), be "damaging to the genetics of the race," "cause sterility," or, indeed, "be carcinogenic over a long period."

In view of the wide demand for and growing use of radioactive iodine in many clinics in this and other countries, it is important to set down the known facts on this subject rather than to rely on rumor or armchair suspicions in determining these cogent considerations in the care of patients who are ill with hyperthyroidism. This is particularly salient, inasmuch as the treatment by means of radioactive iodine is economical of the patient's time and money, and of hospital beds as compared with surgical management; and it is conservative of long periods of invalidism and protracted medical observation required by antithyroid drug therapy. Since I^{131} therapy is virtually 100 per cent efficacious under properly standardized conditions, the deterrents to I^{131} must need be removed as rapidly as is warranted by sound clinical judgment on the basis of accurate information.

Further, since an estimate of over 4,000 patients have already been treated by many observers, [E, F, D, 5, 6] the fears of some of these patients which have been "doctor-induced" must be allayed as soon as possible. Individuals who have been cured of their hyperthyroidism by I^{131}, and who are otherwise happy to have received the therapy, have developed deep-rooted anxieties concerning the "bogey" of damage of "one sort or another" which well meaning, (usually surgically minded) members of the profession have raised. The harm done in such an inadvertent manner has been obviously uncalled for and certainly inhumane when it results from lightly considered and ill advised rumor in place of logically con-

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ceived opinion and adequately collected facts.

Therefore, let us examine the rumors, and what may be their basis, as well. Let us state the pertinent data upon which unbiased advice may be rendered to the hyperthyroid patient who is a candidate for treatment.

There was a period within my own memory in the history of the therapy of this disease during which the choice of method was made upon the basis of mortality statistics. Times have changed to such a degree, as a result of research during the past twenty-five years in this field, that we now think of these matters not in terms of "what are our patients' chances of survival under therapy" but rather in terms of "what are their chances of becoming free of persistence, complication, recurrence or other ill effects of the type of therapy employed." Mortality from hyperthyroidism per se has been erased to the vanishing point except in neglected cases. The death of a thyrotoxic patient is now as inexcusable as is one from diabetes when there is no complicating disease. To state that radioactive iodine therapy may avert all surgery on the thyrotoxic subject is not yet justified, but it is important to realize that many operations now being done on the thyroid are "needless" in the sense that our knowledge to date is sufficient to enable us to avoid many of them!

THEORETICAL CONSIDERATIONS

It is important to understand the theoretical background of the individual forms of therapy for Graves' disease in order to evaluate their relative safety and efficacy for the patient. In general terms, all the methods being used have as their goals the ultimate decrease in the overproduction of thyroid hormone and the avoidance of harmful effects which might be incident to the particular approach used to achieve this primary purpose. Surgery attempts to reduce the total mass of hypersecretory tissue present in these patients by mechanical removal of as much tissue as the skill of the surgeon will allow without damage to the recurrent laryngeal nerves, the parathyroid glandules, and the cardiac and adrenal mechanisms. Technical achievements and proper preparation of the patient by antithyroidal drugs, iodine, general nutritive measures, and cardiac therapy have contributed greatly to the safety of the surgical approach in large centers and in most small hospitals.

However, despite these advances the mortality following subtotal thyroidectomy still varies from 0.5 per cent to 10 per cent, or higher, in individual hospitals in the U. S. A. and Europe. (published and unpublished data.) A survey of these statistics reveals that the deaths have not been limited to the severely ill and aged patients as might have been expected; but deaths have included many mildly toxic cases and individuals of youthful age. By and large, the incidence of thyroid storm as a cause of death has been effectively eliminated by proper preoperative preparation of patients. The deaths have been due in the main to surgical and anesthetic accidents such as bilateral adductor paralysis of the vocal cords, postoperative hemorrhage, tracheal collapse, pulmonary and cardiac infarctions; traumatic shock of irreversible type, sepsis with mediastinitis, parathyroid tetany, air embolism to the brain, and ether convulsions.

Antithyroid medication is presumed to act by interference with the mechanism of fixation of inorganic iodide following conversion in the thyroid to iodine. This results in decrease of thyroglobulin production by imposing a barrier to the enzymatic conversion of iodine into protein-bound iodide. It is evident that this offers the surgeon an excellent aid in the preoperative control of the thyrotoxic element of the disease but it is equally important that the frustrated hyperplasia resultant from exhibition of antithyroid drugs leads to enlarged and markedly vascular glands for the surgeon's mechanical approach. The addition of iodide to the antithyroidally prepared gland has obviated this disadvantage to the surgeon by promotion of the involution of such frustrated hyperplasias. The consequent reduction of vascularity has

restored the surgical advantage of a beautifully controlled thyrotoxic element in the presence of a well involuted gland.

The proponents of the long term therapy of thyrotoxicosis by antithyroid drug administration have found between 60 to 80 per cent of cases in permanent remission after periods of therapy lasting eight to ten months. But close follow-up thereafter has led us personally to the conclusion that the per cent of such antithyroidally induced remissions has decreased with the passage of time to the point where 40 per cent are clinically judged to have taken place on a permanent basis.

Hence we place the efficacy rate of prolonged antithyroidal therapy based on induction of "cure of the disease" somewhat below 35 to 40 per cent. Considering that these results are obtained only with the greatest of persistence on the part of the patient, the doctor, his employers, and with considerable drain upon financial resources, it becomes obvious that while antithyroidal drugs thus far developed have contributed much to our understanding, clinical management, and preoperative preparation of patients with thyrotoxicosis, an ideal nonsurgical treatment for the great majority of patients is not attained by the antithyroidal approach. It is our personal opinion that they aid in control of the havoc of the disease until natural remission occurs (in periods of one to two years), in a manner similar to that in which did iodide therapy without surgery in a limited fashion prior to their discovery. This approach has broadened our knowledge of the mechanism of the disease, but as in the former methods it has not enabled us to eliminate the final common pathway—the inherent cause of the disease has not been discovered or eliminated.

We still need to attack the end result, the hyperactive thyroid gland, which is the end organ through which the various results of the disease are developed. The tendency to remission of thyrotoxicosis is favored by the very adequate control which antithyroidal drugs afford us. They make possible the prolonged psychiatric and nutritional approaches which usually must accompany them for best results. Prolonged therapy is, on the contrary, not possible in patients presenting themselves in a neglected state, with severe and debilitating complications of heart disease, diabetes, and like conditions. Antithyroidal drugs have a definite, though quite low incidence of drug sensitivity and toxicity; or bone marrow, hepatic and adrenal depressant effects which obviate their prolonged use. It is true that rarely does one encounter a case in which the acute antithyroidal effects are not obtainable as was the case with iodide suppression of thyrotoxicosis. Often increases in dosage are needed and prolonged or delayed responses have been seen to occur when, at first, the antithyroidal approach seemed lacking in effect. However, we have, as have others, seen patients whose disease became very prominent after initially good control of same. Suggested increases in dosage at such times have resulted in further amelioration. In our experience subtotal "escape" from the action of antithyroidal drugs has been evident in increasing numbers of patients who have by consequence thereof been referred to us for I^{131} therapy after prolonged disability.

EXPERIENCE WITH EXTERNALLY ADMINISTERED X-RAY THERAPY FOR TOXIC GOITER

In a few quarters treatment by means of external x-irradiation has continued to be practiced. Particularly interesting in this regard were the results of a recently conducted survey reported by Edith H. Quimby and Sydney C. Werner of Columbia University, which was published in the Correspondence Columns of the J. A. M. A., (July 23, 1949; 1046-47). Responses to a detailed questionnaire from 70 radiologists and 31 internists especially interested in treatment of thyroid disease led them to conclude that "if the complication (of cancer) exists following irradiation it is very rare." In discussing the possible late effects of x-irradiation they found only 3 cases in which the history could be interpreted on any valid basis to have suggested that radiation was implicated in the production of cancer in toxic goiter x-ray therapy. Since their survey is estimated to

have encompassed the experience in several thousands of cases over a period of thirty or more years, it would appear that the incidence of cancer in x-ray treated cases was not appreciably higher than is the spontaneous incidence of cancer in toxic goiter glands reported by Pemberton from the Mayo Clinic in cases which had received no radiation.

There is no reason to assume that the tissue ionization produced by a stated number of roentgen equivalents is different,—be they delivered by means of an x-ray tube or by the administration of a beta emitting short, half-lived isotope such as I^{131}. The data accumulated in Quimby and Werner's report with regard to the incidence of malignancy in heavily treated cases by x-ray would argue in favor of a special sensitivity of the skin to x-ray carcinogenesis as opposed to a rather remarkable insensitivity of the hyperplastic and normal thyroid glands of the suman being.

EIGHT YEARS EXPERIENCE WITH RADIOACTIVE IODINE THERAPY

Since 1941 we have observed the salutary effects of radioactive iodine on patients with toxic goiter, but in no instance to date has there been seen the development of any evidence of malignancy of the thyroid. Even in instances in which overdosage has led to the development of myxedema the development of cancer has not occurred. It is reassuring that the x-ray treated cases over a much longer period of time have also been free of this complication.

It might be reasoned by some that by adopting the medical approach,—either prolonged treatment by means of antithyroidal drugs or radioactive iodine, we lay our patients open to the possibility of failure to recognize an early coincidental cancer of the thyroid in a hyperplastic gland. We have borne this clearly in mind and have recommended the removal of any persistent goiter following the successful relief of thyrotoxicosis by either of the two methods.

It might be argued that the radioactive iodine would be effective prophylaxis and preparation for radical care by surgery in such cases of persistent goiter following successful I^{131} therapy and that this extra precautionary step might lead to the earlier detection and more adequate cure of coincidental cancer of the thyroid in these cases.

On the other hand, we have seen the persistence of goiter so frequently after the detoxified state has been reached in the antithyroidal management of thyrotoxic patients that we would predict that over 50 per cent would come to operation even though complete remission be induced in them because of such persistent thyroid masses. The obvious disappointment of such patients on hearing that operation is needed for safety, after one to two years, or longer, of treatment by antithyroidal drugs made them subjectively well, remains a disadvantage of such a program.

THE THERAPEUTIC APPLICATIONS OF RADIOACTIVE IODINE IN THE TREATMENT OF THYROID DISEASES

In July 1948, a conference was held at the Brookhaven National Laboratory, Upton, New York. The proceedings at that conference on the subject of radio iodine have been published as U. S. Report B.N.L. -C -5. This published material summarizes much of the physiologic and laboratory aspects to that date. The concentration of radioactive iodine by goiters, and the mechanisms of antithyroidal medications, as well as the methods for standardization of radioactive iodine administration and therapeutice dosage were covered. Uptake and excretion studies were reported both in benign and malignant conditions of the thyroid and in other endocrinopathies. The reader is referred to this publication as authoritative on these various subjects.

The author had the privilege of acting as chairman of the session devoted to the study and treatment of cases of hyperthyroidism by means of radioactive iodine. The reader is referred to these discussions which are of historical interest,[d] as well as of academic value in relation to this form of treatment.

Succinctly summarized, the session indicated beyond any question of a doubt that *properly assayed dosage and selection of patients with toxic goiter for treatment led to entirely satisfactory cures in the great majority of patients treated by means of*

radioactive iodine. Various methods of approach were utilized, as for instance the oft repeated and small dose (Soley) as opposed to the excessively large dose of other (Chapman) investigators. Somewhere between the two schools lies the ideal dosage for the treatment of hyperthyroid cases. Methods of measurement of both the administered material, and also, the uptake in the thyroid gland being treated have become more standardized and improved so that specific dosage for individual thyroid cases is becoming more clarified.

It was the consensus of the group discussing the problem of the diagnostic use of radio iodine that we had reached a roughly qualitative stage in our knowledge of this aspect of the use of radioactive iodine, but had not approached the point at which one would wish to give up the older forms of diagnostic thyroid testing, such as the basal metabolic rate determination. The chairman predicted, however, that properly conducted turnover rates, utilizing protein-bound I^{131} neosynthesis, might very well turn out to be the most sensitive indicator of thyroid function. This great desideratum cannot be achieved, however, until adequate methods for the determination of protein-bound I^{131} have been worked out. These methods are now becoming available,[8] and it should not be long before a method applied to finger blood utilizing this principle will be a routine measure in all clinics where radioactive iodine is being studied or is available.

The treatment of cancer of the thyroid is based primarily on the same principle as that utilized by Hertz and Roberts in their treatment of thyrotoxicosis by means of radioactive iodine. The situation with regard to the locally static case of carcinoma of the thyroid is clear. The patient should have as radical a removal of tissue as is possible and have subsequent dosage of radioactive iodine administered as long as there is any significant retention of radioactive iodine within the body. It is fair to say that the administration of external irradiation should be considered an outmoded form of treatment wherever radioactive iodine is available. No patient should be discharged as cured of cancer of the thyroid until he has developed total myxedema and in the absence of any metastatic lesion is placed upon thyroid medication and requires it as a permanent form of substitution therapy.

In the case of metastatic cancer of the thyroid with lesions in lung, bone, or other organic metastases, the problem remains one of radioactive iodine in repeated dosage with either local excision of the tumor or massive dosage destruction of the local normal tissue with subsequent repeated dosages of radioactive iodine until no further retention of the material is demonstrated. Since not all tumors are equally avid for radioactive iodine, a very useful procedure has been employed; namely, that of giving thyrotropic hormone or a preliminary treatment of the patient with propylthiouracil for the purpose of promoting increased uptake by lesions which were not originally iodo-receptive.[11]

On the whole the results of radioactive iodine treatment of cancer of the thyroid, while promising, have not indicated any great percentage of cures in the short time in which the procedure has been used.

However, the original patient of S. M. Seidlin[11] of Montefiore Hospital is still alive after having received treatment in 1943 for metastatic thyroid cancer with associated thyrotoxicosis. He has been totally myxedematous and has, as noted by x-rays, remained free of any new lesions. Most of his old ones have decreased to the vanishing point with respect to both radiologic appearance and I^{131} uptake on repeated tracer studies. It seems quite clear that all functioning thyroid tissues which were present in this man have been markedly necrotized by the action of the beta radiations of the radioactive iodine which he has received. Of course, it will take a number of years to demonstrate many such cases as this original one. The author has had contact with this patient since the inception of treatment in 1943, along with Dr. Seidlin, and saw him alive and well as recently as May 1949, a period of six years following

the inception of his therapy by means of radioactive iodine.

DOSAGE CONSIDERATIONS IN THE USE OF RADIOACTIVE IODINE

The primary guide for dosage in the treatment of patients with hyperthyroidism still remains the uptake of radioactive iodine by the thyroid per gram of estimated tissue as checked by external Geiger-Mueller counting and urinary excretion studies. It is our opinion that no patient should be treated without a preliminary tracer study utilizing 100 microcuries of I^{131} for this purpose. The importance of standardizing the dosage on the basis of the tracer behavior of the individual case has been brought out many times in our own and others' experiences; the success or failure of the treatment may be dependent upon such an adequately conducted tracer study preliminary to the therapeutic dose, since the receptivity of the gland for the therapeutic dose is the major consideration with respect to both the size of the dose and its probable effect upon the patient.

Improved methods of measurement of radioactive iodine uptake by the thyroid and other sites of the body are now in development and will be generally available shortly; namely, four-way Geiger counters named by us "Multi-counters" to be used in conjunction with Multi-scalers which read up to 40,000 counts per minute in an accurate and duplicable manner. Urinary studies by the Marinelli technic utilizing a candle type beaker and gamma ray detector have been found to require less time and to be adequate for the usual clinical applications, instead of beta ray counting.

With regard to the dosage for patients with cancer of the thyroid with metastases, each individual case must be treated in accordance with observations in that particular individual. The data so far published do not allow any law to be set down with regard to cancerocidal dosage. However, gross dosage from 100 to total 934 (Seidlin) millicuries have been utilized without undesirable irreversible changes in the patient. Leukopenia should be watched for and also anemia be corrected before aplastic changes have ensued in the bone marrow. The incidence of aplasia of the bone marrow is more suspected than real as there have been no specific reports of death due to any such toxic effect of even these large doses of radioactive iodine.

Nor has the use of these tremendous dosages of radioactive iodine been attended by any renal damage. It is apparent, therefore, that the minor dosages used in patients with thyrotoxicosis are unlikely to be of any renal importance. The extreme dilution of the radio isotope in the urine is a likely explanation of this failure of any renal or bladder damage by the radioactive iodine being excreted through that pathway.

Repeated sperm counts and also the menstrual histories of patients treated by means of radioactive iodine have indicated no damage to either the testicles or the ovary in any functional sense. Normal pregnancies have followed both the treatment of male and female parents with thyrotoxicosis by means of I^{131}.

LONG TERM RESULTS OF TREATMENT OF PATIENTS WITH GRAVES' DISEASE BY RADIOACTIVE IODINE

In the original reports by Hertz and Roberts were included the results obtained to date of March, 1946 for a series of 29 patients treated from March, 1941 to April, 1943, a period of observation covering 3 to 5 years. The results of follow-up conducted as of December 15, 1949 (6-8 years) may be summarized as in Table I.

From Table 1 it can be realized that the operations upon the 5 cases in the original series might have been avoided if confidence in the procedure of I^{130} and I^{131} treatment in 1943-44 had reached its present high level. Retreatment has been needed in 3 patients who received noneffective first dosage; but in no instance was a third treatment found necessary in the original group.

At the dosage level employed in series 1, and with retreatment of the nonoperated "failures," the score for successful treatment of individuals with thyrotoxicosis in this first group has been raised to 24/29. It is fair to assume with our present knowl-

TABLE 1

SIX TO EIGHT YEARS' FOLLOW-UP OF RESULTS ON THE ORIGINAL PATIENTS WITH GRAVES' DISEASE TREATED BY MEANS OF I-130, RADIOACTIVE IODINE MARCH 1941 TO DECEMBER 15, 1949

Clinical Result as of December 15, 1949	Case No.	No. of Cases
Died of rectal cancer (independent of treatment)	22	1
Treated for persistence or (?) recurrence after I^{130} by I^{131}	2, 3, 26	3
Remained well after I^{130}, I^{131} induced remission		17
Treated for recurrence (? one)	3(?)	0
Developed myxedema after long latent period	12	1
Ophthalmopathic (controlled on medical measures) slowly improving on iodide and thyroid	4	1
Operated, myxedema and hypothyroidism	1, 5, 10, 14, 16, 19	6
Total of original series (1941-43) Mass. General Hospital		29

edge of dosage and confidence in our technic that the 5 operated cases might well have responded to a second dose of I^{130} in the absence of operation if such a program had been chosen instead.

The development of evidence of thyroid deficiency after the combined treatment of I^{130} plus operation is of special interest in the light of earlier experiences with combined x-ray and operative treatment reported from the Massachusetts General Hospital by Pittman.

Although 3 cases were retreated for persistence of thyrotoxicosis, it is striking that no instance of true recurrence of the disease occurred. One patient died of independent cancer of the colon (case 22) with no recurrence of thyrotoxicosis. No other patient of the series died either during or since treatment. No complications were encountered. No case has developed cancer of the thyroid, anemia, evidence of renal or hepatic damage. No instance of malignant exophthalmos, tetany, or vocal damage has been encountered in this series or in any other treated by I^{131} and published to the present time.

Chapman and Evans reported in some detail in 1946[12, 18] on the use of higher doses of I^{130}-I^{131} as the sole agent, i.e., no additional iodide therapy during the radiational period. Between May 1943, and March 1945, they treated 22 patients having hyperthyroidism with such increased dosage in the absence of other therapy. They ported that 14 responded well to a single dose; 3 were given 2 doses and 5 required 3 doses. Myxedema occurred in 4 of their cases. Six of their patients developed radiation sickness; and fibrosis of the thyroid was demonstrated by them by biopsy of 2 patients. A comparison of these two series has led us to believe that the dosage was excessive in the latter series and the advantages of control of the disease by iodine administration three to four days after the radioactive dose are sufficiently great to warrant the adoption of this routine as standard, providing that such full iodinization does not modify the thyroid retention of radioactive isotope in any serious manner.

Freedberg, et al[13] under our supervision, reported that such modification of the retention of I^{131} in the thyroid after a therapeutic dose does not occur if the iodinization is accomplished after the third day following the I^{131} therapeutic dose. These authors pointed out that a small peak in urinary excretion of I^{131} does occur following iodinization in this manner, but that this is at the expense of extrathyroidal or total body distributed I^{131}. It is likely that this is an additional advantage to post-I^{131} iodinization rather than a deterrent to the therapy. From the results, now, in the treatment of over 750 cases treated by ourselves and by R. H. Williams, Werner and Quimby, and Soley, it becomes quite evident that the procedure of iodinization does in no way interfere with the desired radiational effects of I^{131}, and that it lends safety to the clinical care of the severely toxic and

TABLE 2

Detailed Analysis of the Clinical Courses of a Representative 13 of 99 Complicated Cases of Graves' Disease with their Special Indications for I^{131} Treatment. (1946-1948)

Initials Age Sex	Diagnosis	Duration years	Complications and/or Special Indications	CLINICAL AND LABORATORY DATA BEFORE I131 TREATMENT: Thyroid Size Est'd. Grams	BMR Level	Circ. Time Secs.	I131 milli-curies	Carrier NaI mgms
E. F.[1] 45 M	Diffuse Toxic Goiter	1	Poor control on iodide Incr'd. size of goiter and exophthalmos on propyl-thiouracil. Refused surgery	75	+45	13	4.0	0.5
R. S.[2] 23 M	Diffuse Toxic Goiter	½	Poor control on iodide	75	+40	14	8.0	0.5
F. A.[3] 44 M	Diffuse Toxic Goiter	½	Incomplete control by propyl and iodide Refused surgery	90	+45	14	8.4	0.5
I. B.[4] 38 F	Diffuse Toxic Goiter	1& ⅓	Refused surgery	100	+40	11	8.0	0.5
Retreatment[5]			Improved but persistent thyrotoxicosis	60	+25	9	6.3	0
M. N. S.[6] 45 M	Diffuse Toxic Goiter	⅓	None	75	+30	....	8.0	0.5
L. D.[7] 45 F	Diffuse Toxic Goiter	1	Psychosis Involutional Melancholia. Drug addiction Refused surgery	40	+40	12	6.4	0.5
C. K.[8] 41	Recurr. Postop. Diffuse Tox. Goiter	1¼	Progressive exophthalmos	90	+45	12	8.0	0.5
S. H.[9] 57 F	Diffuse Toxic Goiter	1	Psychosis-involutional melancholia Hypertension B. P. 200/105	75	+60	14	7.5	0.5
W. P.[10] 35 M	Diffuse Toxic Goiter	3	Bronchial asthma Anxiety neurosis, severe	75	+25	10	6.0	0.5
K. H. 44 M	Diffuse Toxic Goiter	¼	None	45	+30	13	4	0
M. P.[11] 37 F	Recurr. postop. & post x-ray Diffuse Toxic Goiter	6	Failure of control by x-radiation and by iodides	75	+25	16	4	0.5
S. B.[12] 27 F	Diffuse Toxic Goiter	¼	Healed tuberculosis Sensitivity to propylthiouracil	60	+26	11	4	0
F. F.[13] 56 F	Recurr. postop. Diffuse Toxic Goiter	22	4 thyroidectomies. Persistent after x-ray therapy. Granulo-cytopenia following thiouracil. Incomplete relief by propyl-thiouracil and iodide	60	+30	8	12	0

COMMENTS

.... = not obtained or unknown
+0 = none
r.e.p.* = roentgen equivalent physical

[1]11months after I^{131} BMR +1. Euthyroid. Exophthalmos improved.

[2]Transient myxedema, 14 months after I^{131}, off thyroid 6 weeks, BMR –21, serum cholesterol 276 mgm. %.

[3]BMR +3 1 year after I^{131}. Off medication 9 months.

[4]Improved but persistent thyrotoxicosis 5 months after I^{131}. Retreated 11 months later. (Protein Bound Iodine 15.8. gamma %).

[5]Euthyroid (eight weeks after second treatment).

[6]Lost to follow-up. L. M. D. reports that patient is well.

TABLE 2

Detailed Analysis of the Clinical Courses of a Representative 13 of 99 Complicated Cases of Graves' Disease with their Special Indications for I^{131} Treatment. (1946-1948)

	TREATMENT AFTER I^{131}							EVALUATION 6 MONTHS AFTER I^{131}			
			Propylthiouracil								
Urine I^{131} Excretion in 72 hrs. % Dose	Estimated Radiation Delivered to Thyroid r. e. p.*	Stable Begun Days After I^{131}	Iodide Weeks Used	Begun Days After I^{131}	Weeks Used	Clinical Status	BMR Level	Circ. Time (Secs.)	Wgt. Gain Lbs.	Thyroid
61	2600	1	6	0	0	Euthyroid	+5	16	24	Normal
63	4600	1	11	0	0	Myxedema	On Thyroid	26	35	Normal
....	.....	1	12	0	0	Euthyroid	.....	....	30	.....
....	.....	1	10	0	0	Persistent Thyrotoxicosis	+20	14	0	60
14	10,600	0	0	0	0	Euthyroid	.....	....	8	Normal
....	.....	1	1	0	0	Euthyroid	.....	....	....	Normal
....	.....	1	8	0	0	Euthyroid	+7	....	22	.....
25	7800	3	12	0	0	Euthyroid	+5	18	17	Normal
25	8800	0	0	0	0	Euthyroid	+4	16	13	Normal
16	7900	0	0	0	0	Euthyroid	–3	17	8	Normal
28	7500	3	7	0	0	Euthyroid	.....	....	....	.....
5.41	2900	3	5	0	0	Euthyroid	+8	17	0	Palpable Tissue Persists
21	6200	0	0	0	0	Myxedema	.....	30	12	Normal
25	17,500	8	....	0	0	Euthyroid	.....	15	15	Normal

COMMENTS

[7]Hypometabolism without clinical evidence of myxedema. 9 months after I^{131} BMR –20. Cholesterol 285 mg. % circulation.

[8]Progressive decrease of exophthalmos. 8½ months after I^{131}; patient euthyroid.

[9]9 months after I^{131} BMR –22. Serum cholesterol 716 mgm. %. Clinically myxedematous. B. P. 200/110.

[10]Euthyroid 9 months after I^{131}. Exophthalmos completely disappeared.

[11](1) 4 weeks off iodide. (2) Marked decrease in size of goiter. Left lobe still palpable. Repeat I^{131} tracer showed 26% excretion in 72 hours.

[12]Euthyroid (BMR +0) with no palpable thyroid 4 weeks after I^{131} without added therapy. Rapid response.

[13]Myxedema—on permanent thyroid medication. (9 months after I^{131} therapy).

desperately ill cardiac and diabetic subjects with thyrotoxicosis.

The most complicated and debilitated patients have been handled with I^{131}. Patients with serious complications such as colostomy, congestive heart failure, Addison's disease, renal disease, multiple antecedent operations, drug sensitivities to iodide, and propylthiouracil, as well as patients with severe infections, have responded favorably to I^{131} therapy followed by routine iodinization (see Table 2)). Detailed reports of these subsequent series of patients are available.[E, 16, 17]. In no instance was it thought that iodinization contributed negatively to the care of the patients in such instances in which iodinization was not carried out prior to the seventy-two hours following the therapeutic I^{131} dose.

In emergency cases it is our view that, if allowance is made for the possibility of a moderate loss of I^{131} by iodinization during the first forty-eight hours following I^{131}, by adjustment of the dosage of I^{131} upwards it is permissible to carry out full iodinization relatively early in order to bring the patient's disease under as rapid control as is possible by any known technic.

We are now in a position to caution against certain pitfalls in the practical management of patients with I^{131}. We have discovered a few of these by the experience of analyzing the new failures of our own and others' cases.

A priori and by actual experience we agree with Werner, who states that the most common cause of failure of a single dosage to remit the disease is inadequate dosage. Our present dosage scheme calls for 200 to 250 uC/gram of estimated thyroid weight. By clinical practice it is possible to calibrate one's palpation of the thyroid, as has been shown by Soley, to a fair accuracy in such estimation of thyroid weight. Werner's models are also helpful adjuvants in such self education in this important aspect of thyroid examination. Unless the thyroid is of inordinate size, i.e., over seven to ten times normal, it is unlikely that over 12 to 15 MC. total dosage will be required. We have had cases in which response has occurred to as little as 1 MC. for an impalpable gland, (probably less than 25 grams); and yet we have seen failure on the first dose from as much as 12 MC. for a patient with a 90 to 100 gram goiter. In such instances we believe it better to err on the side of low dosage and to depend upon the use of a second dose, if it proves to be needed in follow-up.

In our present state of knowledge and with improved equipment soon to be made generally available, it should be made a rule that no patient with thyrotoxicosis should be treated with I^{131} without a preliminary tracer dose of 100 uC. and determinations of both the thyroid uptake and urinary excretions over a period of forty-eight to seventy-two hours.

We have found a close correspondence between the handling of tracer and therapeutice doses by patients in our large series. The importance of these preliminary tracer studies is heightened by certain instances in which patients were taking iodinized salt, kelp or cough mixtures prior to presentation for I^{131} treatment. Early studies in both animal and man indicated the importance of having the systems free of extraneous iodide ingestion prior to therapeutic uptake of I^{131}.[1-4]

In one case 3 doses of I^{131} had been administered at another clinic without tracer or excretion studies. This patient responded very little to those doses and presented herself to us for study because of this alleged resistance to I^{131} treatment. We performed a tracer study and found her gland unreceptive to I^{131}. On analysis it was elicited that for over four years she had used iodized salt regularly and had been continuously on this source of iodide during her entire I^{131} trials. When iodized salt was stopped for a month and a repeat tracer dose given it was discovered that her uptake was consistent with her moderate thyrotoxicosis; and a subsequent therapeutic dose was fully effective when taken on an "empty thyroid," i.e., free of extraneous iodide.

Patients who have received the antithyroidal agents, such as thiouracil and pro-

pylthiouracil, alone without iodides offer less of a problem than those previously on iodide medication from the above point of view. It is, however, imperative that the goiterogen be stopped for at least three days prior to the tracer or therapeutic dose as patients who are under the influence of these drugs do not display characteristic uptakes of I^{131} by their goiters. This, in part, has accounted for an additional number of "failures" on I^{131} therapy and re-emphasizes the value of the preliminary tracer study as a control on optimal time for treatment.

Tracer studies also afford an added bit of information in guiding the dosage to be used in individual cases for therapy.[6] Although Werner's results indicate that the tracer behavior of I^{131} in cases of atypical hyperthyroidism is characteristic, we have not been impressed with the utility of thyroid uptake studies or excretion studies as diagnostic aids in borderline or early cases of toxic goiter. Current studies, however, give promise that the turnover rate of I^{131} into protein-bound I^{131} in the blood after a tracer dose may be made discretely dependable for this purpose; particularly, when microtechnics on finger blood are fully developed. This is work in progress at this time.

Complications of I^{131} therapy have continued to be few and mild in character. Roughly 10 patients in 100 develop myxedema. Of these 10, only one has proved to be permanent in character; that is, one case in 100 requires post-I^{131} therapy of a permanent sort in our hands. That myxedema may eventually be entirely avoided seems likely when accuracy of dosage is further improved and as the fractional treatment of patients becomes more utilized.

Minor tenderness of the gland, slight cough and, rarely, slight exacerbation of thyrotoxicosis have been experienced in our group; but no cases have given us cause for concern with the ready use of local measures and the early use of post-I^{131} iodinization. We have not encountered leukopenia or anemia; no fever or radiation sickness has been noted in our more moderate dosage group.

Repeated renal studies over a period of eight years have revealed no evidence of either acute or chronic long time effects upon the kidneys of our subjects. Fertility has not been decreased in our series; we now have 16 babies born to 11 mothers and 3 fathers who have undergone I^{131} treatment for thyrotoxicosis previous to conception. One of these mothers has had 3 separate pregnancies (normal, full term fetuses at birth). None of the 16 babies has shown evidence of cretinism or congenital defects.

There are still a few observers who raise the question as to whether I^{131} treatment of thyrotoxic patients is going to result in cancer of the thyroid as one of the delayed effects of concentrated radiation delivered to the organ during such therapy. Calculations made by Robley D. Evans and reported by Earle M. Chapman at the Brookhaven National Laboratories Conference on Radio-Isotopes in Biology and Medicine (*q.v.*) give valid assurances that this is a minor likelihood. Further reassurance is given from the long term experience in many lands (over a period of thirty years) of the nondevelopment of any significant number of cases of cancer of the thyroid following the more drastic treatment by intensive x-ray treatment over the thyroid gland for this and other conditions of the cervical region.[14]

A similar answer can be given to those who object to I^{131} therapy on the basis that the genetics of the race will be altered by this method of treatment. Calculations of the probability of such genetic changes taking place as a result of the minor deposit of I^{131} in the testicles or ovaries of our patients render such objections remote, indeed.[15] Actuality of fertility has been proved repeatedly following I^{131} therapy in our series.

That these therapeutic effects can now be regularly obtained by proper dosage, consideration of the underlying principles of the type of therapy and due care to perform proper standardization of the approach by preliminary tracer studies, becomes more

evident as the method enjoys greater application.[8-10,13]

There have been excellent accounts of the results obtained by others using I^{130} and I^{131} in treatment: Notably by Werner, Schmidt, and Quimby; and Soley, Chapman, Skanse, Evans and Printzmetal. In essence, these authors and others conclude, as do we, that I^{131} therapy of hyperthyroidism is effective, safe to the patient and to the doctor with proper precautions set out by the Atomic Energy Commission, and that it is free of some of the drawbacks of either the surgical or other medical approaches to the problem.

The contribution of radioactive iodine studies to the field of thyroid physiology, chemistry and pathology continues at a rapid rate.[C] Of these we shall plan to report in full detail on some other occasion. At this time we wish to emphasize the importance of fundamental studies on the subject: "How does I^{131} exert its characteristic action on the hyperplastic gland of Graves' disease?" Inherent in this mechanism are the secrets of cellular proliferation, and enzyme action in the cells. The hope of understanding these is closer as applied to the problems of this disease than in many other fields of medicine or biology, because of the accuracy of the measurements that can be applied and because of the clear-cut effects with which we must now deal.[7]

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MYOCARDITIS*

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SHREVEPORT

Fiedler, in 1889, described a type of myocarditis which he called isolated myocarditis since there was involvement of the myocardium without involvement of the endocardium or pericardium and there was no associated disease which might be the cause. Scott and Saphir,[1] in 1929, reported 2 cases and collected 36 cases in the literature. Simon and Wolpaw,[2] in 1935, collected 10 additional cases and reported 2 others. Other authors have reported individual cases.

Isolated myocarditis or Fiedler's myocarditis denotes a more or less diffuse inflammatory change in the myocardium of wide variety and varied etiology; the principal characteristics they have in common are isolated involvement of the myocardium by a nonspecific lesion without inflammatory changes in the endocardium and pericardium.

It may occur at any age having been reported from 10 months to 55 years but more commonly between 15 and 30 years. The diagnosis of myocarditis usually is not made and frequently heart disease is not suspected. The heart usually is enlarged, the pulse rapid, the arterial pressure low, and murmurs may or may not be present. Death frequently occurs suddenly as in 9 of the 13 reported by Saphir. Generally the symptoms are of short duration without evidence or history of previous infection.

In a review of the material at Shreveport Charity Hospital, we have collected 4 cases conforming to the clinical characteristics and on whom the pathological diagnosis was isolated or Fiedler's myocarditis. They form the basis of this report. The ages were 21, 15, 40 and 22. They were all in colored patients, 1 female and 3 males. The duration of symptoms prior to admission varied from fourteen to two hundred and seventy days. In 2 cases there was a history of recent respiratory infection. The important symptoms were dyspnea, palpitation, edema, pain, and nausea and vomiting. Dyspnea was present in every case, varying from mild with exertion to orthopnea, and was usually progressive. Edema was present in every case, usually moderate, of the ankles and legs. Pain was a symptom in 2 cases, usually in the upper abdomen, but in 1 case was a severe cramping pain rather generalized over the abdomen.

The hearts were all enlarged, confirmed by x-ray in 2 cases; the rate rapid, varying from 100 to 136. Generally there was a simple sinus tachycardia, though premature beats were frequent and a gallop rhythm was noted in 2 cases. The blood pressure was not obviously disturbed, the systolic pressure varying from 90 to 150 and the diastolic from 80 to 100. The lowest pressure recorded was 90 systolic and 60 diastolic, and the highest 150 systolic and 92 diastolic. Pulmonary emboli were diagnosed clinically in 2 cases. The stay in the hospital varied from six hours to fifty-three days. Two patients had two admissions, both expiring on the second admission. They all died suddenly and at the time of death 2 complained of substernal pain and 2 presented the picture of shock. The diagnosis of nonspecific myocarditis was made in 2 cases.

Electrocardiograms were obtained in 3 cases. The changes varied and certainly were not characteristic. Low voltage of the QRS complexes, inversion of the T waves in one or more leads, and an absent or low R wave in CF/4 were noted. One patient had a PR interval of 0.22 and left bundle branch block.

At autopsy all the hearts were enlarged, weighing 540, 440, 520, and 560 grams with evident dilatation and hypertrophy.

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Gross scarring was noted in 3 cases. Mural thrombi were present in 2 cases and pulmonary infarcts in 3. Pleural effusion was present in 4 cases and ascites in 1.

In general the microscopic picture in all 4 of the cases showed necrobiosis and sarcolysis of the myofibrils. In some places the nuclei of the myofibrils were clumped together and the sarcoplasm vacuolated. The inflammatory exudate consisted chiefly of lymphocytes with occasional polymorphonuclear leukocytes. Fibrosis and scarring of the myofibrils constituted a prominent feature in 1 or 2 of the cases. Such areas were judged to represent older or healed lesions. The epicardium was spared in the sections and very little if any changes were noted in the coronary arterioles.

DISCUSSION

Pathology: Microscopically the myocardium presents a fairly characteristic picture. There is diffuse infiltration of the interstitial tissue by lymphocytes, mononuclear cells, and to a lesser degree, polymorphonuclear leukocytes, eosinophiles, and plasma cells. Small areas of necrosis are occasionally found in the muscle fibers and fibroblasts and new blood vessels may be seen. The same heart may show a picture varying from that of cellular infiltration to that of various stages of fibrosis. The endocardium and pericardium are usually not involved. The remainder of the body shows changes due to the passive congestion and infarcts secondary to mural thrombi.

Covey,[3] in a report of his case, states that the general picture is that of muscle cell degeneration with infiltration of a variety of cells, chiefly lymphoid, plasma, and polymorphonuclear cells. In the oldest lesion, fibroblastic proliferation is seen. Many areas are to be seen in which the only lesion is a sharply localized necrosis of a muscle fiber which disintegrates. After the necrosis is well established cellular infiltration begins in the surrounding interstitial tissue. Still later proliferation of fibroblasts is seen. In summary he states that the process is one of necrobiosis of muscle cells, followed by cellular infiltration and finally by replacement by fibrous connective tissue. Saphir[4] mentions two distinct types; one characterized by the presence of granulomatous lesions and the other by a more diffuse type of inflammation without the formation of granulomas, though the so-called giant cells may be present.

Hansmann and Schenken[5] reported a case and suggested that the sequence of events in the development of the lesion was (1) injury of muscle fibers; (2) fracture of muscle fibers with hemorrhage, (3) leukocytic infiltration, (4) clearing away of fragments of tissue by macrophages, and (5) scarring.

Marcuse[6] has reported on 36 cases of non-specific myocarditis. He states that there has been considerable discussion as to whether or not myocarditis is primarily of interstitial nature. In his series of cases the inflammatory process seemed to have originated in the interstitial connective tissue and any involvement of the muscle fibers was apparently of secondary nature.

Etiology: As the name implies the exact cause of nonspecific myocarditis is not known. We may, though, speculate on the possible cause or causes that may be active in the disease. Magner,[7] in his report of a case, discussed the possible etiologic factors, such as Hutchinson-Boeck's disease, syphilitic myocarditis, and tuberculosis; none of which seemed applicable. He was not in accord with the idea that isolated myocarditis was a distinct disease entity due to a specific cause, but rather held it might be due to a variety of causes.

Saphir[4] discussed the etiology of the two types; granulomatous and diffuse, suggesting that the granulomatous type might be caused by a specific organism of unknown variety or one that was not found at the time of examination. Tuberculosis or syphilis might be the cause. Blastomyces and the pasteurella of tularemia were also mentioned as possible agents. A variety of causes were mentioned for the diffuse type, such as burns, infections of the upper respiratory tract, influenza, toxemias and injuries of the myocardium brought

about by such chemicals as sparteine and epinephrine.

French and Weller[8] reported finding an interstitial myocarditis rich in eosinophiles in 126 patients whose sole common factor was that one or more of the sulfonamide drugs had been administered shortly before death. This factor could not be invoked in the cases under discussion but may be the responsible factor in other cases.

Helwig and Schmidt, according to Robinson and Queen,[9] have described a filter-passing agent producing myocarditis in anthropoid apes and small animals obtained from the hydrothorax fluid of a gibbon which dropped dead, and the hydrothorax fluid and spleen of a chimpanzee dying very suddenly. The filter-passing agent was passed through a series of 122 mice and with rare exceptions regularly produced paralysis followed by death or apparent recovery after a week or two. At necropsy interstitial myocarditis was found in almost all animals. In some it was very severe; in others limited to small foci of necrosis and inflammation. Such an agent has not been previously described and strongly suggests that isolated myocarditis of humans may well be a virus disease also.

Saphir[10] in a discussion of virus myocarditis, points out that myocarditis occurring in man in various virus diseases, is being reported more and more frequently. From a clinical standpoint importance should be attached to virus myocarditis connected with the following: epidemic infectious hepatitis, infectious mononucleosis, yellow fever, varicella, dengue, poliomyelitis, atypical primary pneumonia, rubeola, measles, mumps, and influenza-like infections. Owing to the benign character of most of these diseases, the myocardial involvement is overlooked.

Covey[3] mentions the fact that the only immediately preceding or accompanying acute or infectious disease which has been reported in conjunction with Fiedler's myocarditis in more than one instance is influenza or acute upper respiratory infection. He states that in 6 of the 14 cases in which the preceding disease or possible etiologic agent has been mentioned, this was at least an influenza-like infection. In the present group of cases an upper respiratory infection was mentioned at least twice. Covey states that the life history of the disease as depicted in the myocardium itself is suggestive of explosive cell destruction by a virus, followed by an inflammatory type of lesion and finally, beginning fibrosis. He states that his study suggests that Fiedler's myocarditis is a virus infection of the heart muscle.

Schenken and Heibner[11] reported a case of diffuse isolated myocarditis from which they isolated the *streptococcus hemolyticus* in pure culture from the heart muscle. They state that Rindfleisch isolated the *staphylococcus citreus* from the myocardium in one case and feel that if the myocardium were cultured in each case, specific organisms would be found more often.

Candel and Wheelock[12] have reported 11 patients on whom the clinical diagnosis of acute myocarditis was made. It followed peritonsillar abscess, lobar pneumonia, primary atypical pneumonia, scarlet fever, infectious mononucleosis, typhus fever, acute recurrent gonococcal arthritis, and urethral stricture with retention and cystitis. They point out that the 11 cases had the following characteristics in common:

(a) Each was associated with an acute infectious disease and further that *Case 3* was seen two weeks after discharge from treatment for pneumonia and *Case 5* had an acute recurrence of gonococcal arthritis.

(b) Each showed significant electrocardiographic changes.

(c) Each had a rapid erythrocyte sedimentation rate even when the primary disease had abated (with the exception of *Case 8*, where no erythrocyte sedimentation rate was done).

It seems quite possible that such cases as these are far more frequent than suspected. Severe involvement of the myocardium or repeated attacks resulting in extensive damage to the myocardium could produce the picture which we know as acute nonspecific myocarditis.

Gore and Saphir[13] have reported a case

of myocarditis associated with acute nasopharyngitis and acute tonsillitis. Saphir also collected 35 cases of nonrheumatic myocarditis associated with upper respiratory infections; 12 with acute tonsillitis and 23 with acute nasopharyngitis. He[14] further reports that 75 per cent of the cases of myocarditis in a series of 1,402 collected at the Army Institute of Pathology, were nonrheumatic and nondiphthertic. Saphir[4] again collected 240 cases of myocarditis in 5,626 routine autopsies, an incidence of 4.25 per cent. He finally refers to 5 nonfatal cases following acute tonsillitis reported by Scherf,[15] who estimates the occurrence of acute myocarditis complicating such infections at 10 to 15 per cent.

Marcuse[6] in a review of the possible causes noted in the literature, lists the following: bacterial infections, virus, sensitization to various substances, drugs causing increased heart action, and dietary deficiency such as general malnutrition, potassium deficiency, and combined deficiency of potassium and vitamin B. In the 36 cases analyzed by Marcuse, inflammatory lesions in organs other than the heart were found in 35 of the 36 cases. Some of the clinical records contained evidence of an infectious process that was present some time prior to the onset of the final illness. This fact, together with the difficulty of demonstrating any organism in the heart, suggests that the bacteria exert their influence from a distant focus by means of their toxins rather than by direct invasion of the myocardium.

The first patient in this series is interesting from the possible relationship of the thyroid. She had been followed very closely and the diagnosis of isolated myocarditis had been generally agreed upon. She expired rather suddenly in a state of shock. At autopsy microscopic evidence of hyperthyroidism was found, as well as extensive myocardial damage in a heart weighing 540 grams. Microscopically the heart was classified as myocarditis such as seen in the group classified as isolated myocarditis. The pathologists, Dr. Mathews and Dr. Cummings, made this comment:

"In view of the fact that the coronary arteries showed no significant changes grossly or microscopically the etiology of the lesion present in the heart cannot be determined. In our experience, we have not encountered such lesions of the myocardium in hyperthyroidism. Therefore we believe that one would be justified in classifying the heart lesion as chronic interstitial myocarditis of indeterminate origin."

In one of the cases reported by Helwig and Wilhelmy[16] the thyroid showed microscopic changes of moderate toxicity. They state that the relationship of a moderately toxic thyroid gland should be considered. Gouley, McMillan and Bellet[17] referred to the possible relationship of thyrotoxicosis to idiopathic myocardial degeneration following pregnancy, but did not think it was a factor in their cases. Perhaps a more careful and thorough study of the thyroid in these cases would be enlightening.

The possible relationship of isolated myocarditis to infections particularly of the respiratory tract is illustrated by the following case. W. A., a colored male, 40 years of age, was admitted December 23, 1945, and expired December 24, 1945. His complaint was vomiting and he did not appear acutely ill. The blood pressure was 130 systolic and 80 diastolic, the rhythm regullar, no murmurs and no evident enlargement. He expired suddenly. The pathologist made this note on his report:

"This patient appears to have died of acute heart failure which I believe may be explained by the acute interstitial myocarditis. I should call this Fiedler's myocarditis were it not for the associated interstitial pneumonia, which is consistent with viral origin. However, Golden in a study on pathologic material from 80 cases of atypical pneumonia of unknown etiology, did not describe associated cardiac lesions."

This type of myocarditis has also been seen in Still's disease, as illustrated by the case of G. S., colored male, 16 years of age, admitted to the hospital June 23, 1942, and expired July 21, 1942. His chief complaint was dyspnea and edema. Since 1935 he had been confined to his bed and chair because of stiffness and contractures of the legs. The heart was enlarged on physical and x-ray examination, the rhythm regular and no murmurs were heard. The electrocardiogram showed a sinus tachycardia with

low voltage, QRS complexes and T waves. He was afebrile throughout his hospital stay, expiring suddenly on July 21, 1942. At autopsy the heart was dilated and hypertrophied weighing 380 grams. The valves were normal.

The microscopic report by Drs. Mathews and Cummings was as follows:

Heart—The most conspicuous features of the sections of the left ventricle are those of diffuse coagulation necrosis. The myofibrils persist as shadow-like fibers which are poorly stained and the nuclei are pyknotic. In places there is scarring and fibrosis of the myocardium. Other areas show degeneration of the myofibrils without loss of the nuclei. Here the nuclei are prominent and are surrounded by a vacuolated appearance due to the degenerative changes of the myofibrils. In such areas there is very little evidence of inflammatory change, there being only a slight infiltration of lymphocytes. Sections of the right ventricle show diffuse chronic inflammatory changes of the endocardium and adjacent papillary muscles. The exudate consists of lymphocytes, monocytes, occasional plasma cells and polymorphonuclear leukocytes. Adherent to the endocardium in this area is a mural thrombus that shows evidence of organization. Other sections of the right ventricle show areas of coagulation necrosis with minimal fibrosis and inflammatory changes similar to the lesions in the left ventricle. Sections of the epicardium show slight lymphocytic and monocytic infiltration, congestion and what appears to be edema of the fibrofatty tissue. Aschoff bodies could not be found in a fairly liberal number of sections of the myocardium. Also special stains failed to reveal the etiology of the lesions. However, in consideration of the clinical data and gross anatomical findings, one would not be justified in classifying the myocardial lesions as chronic isolated myocarditis. Definite etiology may have been established if more sections were available for study.

Then there is the relationship of isolated myocarditis to the myocarditis associated with pregnancy. This is illustrated by the case of a 17 year old colored girl admitted to the hospital one month after delivery. Her chief complaints were dyspnea, pain in right side of the chest, hemoptysis, and palpitation. The heart was enlarged, the rate rapid, no murmurs were heard, and the blood pressure was 100 systolic and 80 diastolic. The electrocardiogram showed a rate of 100, with an inverted T-4, isoelectric T1, 2, 3 and moderate right axis deviation. At autopsy the heart was enlarged, weighing 500 grams and there were no organic valvular lesions. The microscopic description by Drs. Mathews and Cummings was:

"Heart: Sections of the left ventricle reveal diffuse focal areas of coagulation necrosis and sarcolysis of the myofibrils associated with microscopic foci of hemorrhage, lymphocytic and monocytic infiltration. In addition the nuclei of the degenerating myofibril are prominent in such areas. Occasionally, similar lesions are found in the myocardium of the right ventricle. Fibrosis and scarring of the myocardium is minimal in these sections. This type of myocardial lesion has been described as myocarditis of pregnancy (Amer. J. Med. Sc. 194:185, 1937)."

Other illustrations might be cited to show that lesions microscopically similar to that described for Fiedler's myocarditis may be found associated with a variety of infections and even with pregnancy. If there is an associated infection, the myocarditis is attributed to it either through direct action of the organism or its toxin. But in the case of pregnancy and Fiedler's myocarditis we do not know or are unable to assign a causative agent which seems to be the essential difference between them.

We probably should be more conscious of acute myocarditis, especially so since it has been shown clinically to be associated with relatively mild infection of the tonsils, nasopharynx, and lungs. Unusual weakness, rapid pulse, and minor changes on the electrocardiogram should be sufficient reason to go slow in the convalescence ond perhaps avoid more serious trouble later.

There are two noteworthy features of these cases apart from their etiological considerations. First, they all have large hearts. This has been noted in practically every case reported in the literature. Frequently, the hearts have been large and yet the patient without symptoms until shortly before death. The basis of the hypertrophy as well as the dilatation must be the myocardial disease or damage. How long this process has been progressing cannot be accurately determined.

The second interesting feature is that they have frequently died suddenly and often with substernal pain. Sudden death has been noted by various authors. Hansmann and Schenken noted sudden death in 13 of the 18 cases compiled. The pain has

all the characteristics of coronary insufficiency. It is reasonable to assume that with large hearts the coronary bed has not kept pace with the myocardial dilatation and hypertrophy. As a result, even though the coronary vessels are fully patent, they are not yet adequate. This may be the basis for the continued dilatation, failure, and sudden death with substernal pain. It is also possible that the sudden death is due to asystole or the development of an abnormal rhythm such as ventricular tachycardia and/or ventricular fibrillation.

CASE REPORTS

Case No. 1: N. B., 21 year old colored female, admitted to Shreveport Charity Hospital February 3, 1950, and expired suddenly March 27, 1950. Her chief complaints were edema of the hands and feet, orthopnea, dyspnea, and tachycardia with slight exertion, all having been present only two weeks. Three weeks prior to admission she had a sore throat and upper respiratory infection. There had been frequent bouts of tonsillitis with no history suggesting rheumatic manifestations. There had been one pregnancy two years before admission.

Physical examination showed the blood pressure to be 150/92 with a pulse rate of 136 without arrhythmia. The tonsils were quite large but not actively inflamed. The heart was thought to be somewhat enlarged and without murmurs. The abdomen was distended moderately, a fluid wave was present and the area of liver dullness was increased. There was 4 plus pedal and 2 plus arm edema.

The initial laboratory reports showed a trace of albumin in the urine; hemoglobin of 9 grams or 58 per cent, 4.2 million red blood cells, 11,650 white blood cells, with 89 per cent segmented neutrophiles, 8 per cent lymphocytes, 3 per cent monocytes. Agglutination tests for typhoid, paratyphoid, typhus, and brucellosis were negative. Blood serology and two blood cultures were negative and a sedimentation rate was not abnormal. Later blood counts showed a rising leukocytosis with no further shift to the left. The albumin globulin ratio was 1:1.03.

On February 8, 1950, twenty-five days after admission, the patient suddenly went into shock with profuse sweating, dyspnea, unobtainable blood pressure and tachycardia with a rate of 170. There was no chest pain, cough, or hemoptysis, and no rales were heard. The electrocardiographic findings were compatible with a pulmonary embolus and an x-ray of the chest showed two wedge shaped areas of density in the right lung. The patient was given heparin and dicumarol with adequate prothrombin response. The tachycardia continued for four weeks along with nausea and vomiting. The electrocardiogram at this time showed evidence of pericarditis. Upon receiving quinidine sulfate, gr. 3 four times a day, the rate slowed to 80. After four days of improvement, on March 27, 1950, she was suddenly seized with coughing, gasping inspirations, and quickly expired.

The postmortem examination revealed bilateral hydrothorax, over 1 liter, to each side, with the right lung almost completely collapsed. There was an infarction of the right lung, but no embolus or antemortem thrombus could be demonstrated in the pulmonary vessels. The heart weighed 540 grams being large, ovoid, and quite flabby. The valves appeared normal and without vegetations. Thrombi were present in both ventricles and the left auricle. There was an infarcted area 1½ cm. in diameter at the apex near the interventricular septum. There was a patent foramen ovale 1½ cm. in diameter incompletely closed by a movable septum. There was chronic passive congestion of the lungs, liver, kidneys and spleen.

Gross examination showed the thyroid gland weighed 28 grams and was moderately firm in consistency. The surface was smooth and the capsule appeared tense. The cut surfaces appeared compact, finely lobulate and reddish brown in color. There were small irregularly shaped greyish white areas scattered throughout the lateral lobes and isthmus. These areas measured as much as 3 mm. in their greatest dimensions. No nodules were present on multiple parallel sections.

Microscopic examination of the thyroid: There was diffuse hyperplasia of the acini which varied in size and shape. The majority of the acini were small and lined by high cuboidal cells and contained very little colloidal substance. Other acini were dilated showing budding of the epithelium and were lined by low cuboidal epithelium. There was an abundance of colloidal substance in these acini. Scattered in the stromal elements were focal areas of lymphocytic infiltration.

Microscopic examination of the heart: There was moderately diffuse coagulation necrosis of the deeper zone of the myocardium adjacent to the endocardium. In association with the necrosis were areas of hemorrhage and lymphocytic infiltration. In addition to these areas were small microscopic foci of microbiosis of the myocardial fibrils associated with lymphocytic infiltration. Occasional polymorphonuclear leukocytes were present but the predominating inflammatory cells were lymphocytes. The adjacent and surrounding myofibrils were poorly stained, showing fragmentation and loss of cross striation. In other areas there was separation of the intercolated discs. In addition, there were mononuclear phagocytic cells containing granular golden brown pigment in the areas showing hemorrhage.

Adhered to the papillary muscles were mural thrombi. Other sections of the myocardium showed fibrous scarring, but this was not a prominent feature of the lesions, so far as these sec-

tions showed. The small blood vessels of the myocardium showed no significant changes.

Comment: In view of the fact that the coronary arteries showed no significant changes grossly or microscopically, the etiology of the lesions present in the heart cannot be determined. In our experience, we have not encountered such lesions of the myocardium in hyperthyroidism. Therefore, we believe that one would be justified in classifying the heart lesions as chronic interstitial myocarditis of indeterminate origin.

Case No. 2: D. H., colored male, age 15 years, entered the Shreveport Charity Hospital July 26, 1948, and died suddenly July 29, 1948. The chief complaint was swelling of the feet and abdomen. There was no history of antecedent upper respiratory infection, rheumatic manifestations, or any illness other than measles in 1940. The present illness began almost two months before admission on June 2, 1948, with intermittent generalized abdominal pain accompanied by dyspnea. On June 22, 1948, he developed edema of the legs, abdomen, and eyelids, with orthopnea and a chronic cough productive of frothy sputum. Just prior to admission, edema of the extremities was so severe as to produce vesication. The patient's father punctured the vesicles with holly leaves producing weeping over the feet and legs.

On physical examination the blood pressure was 90/80, pulse rate 128, and respiratory rate 28. Orthopnea and marked generalized edema including ascites with a fluid wave were present. The skin and sclerae were moderately icteric. There were dullness and moist rales present in both lung bases. The heart was enlarged with the PMI in the sixth intercostal space at the anterior axillary line. No murmurs were heard. The liver was palpable three fingers below the costal border and tender. The venous pressure was 275 mm. of water. The urine was not remarkable. There was a leukocyte count of 9,200 with a shift to the left; there were only 9.5 grams of hemoglobin and a sickle cell preparation showed no sickling. Upon admission he was digitalized, placed on oxygen and given salyrgan intramuscularly. Early the second morning his respirations suddenly became gasping in nature and he quickly expired.

Autopsy revealed moderate bilateral pleural effusion, multiple small recent infarcts in the left lung and a large infarct involving the entire lower lobe of the right lung. The heart weighed 440 grams, was large and ovoid with all chambers hugely dilated. The myocardium was pale red and wet without areas of gross scarring. The valves were free from organic lesions. The liver was enlarged and showed the typical nutmeg appearance on cut section. There was thrombosis of both femoral veins. Microscopic study of the heart showed in both ventricles hypertrophy and generally loosely arranged muscle fibers. Spindle cells (fibrocytes) were prominent in the spaces between the muscle fibers, with some areas of interstitial fibrosis. The inner portion of the right ventricle presented an infarcted appearance with almost complete disappearance of the musculature and replacement by hemorrhagic granulation tissue. Similar focal areas and small foci of sarcolysis were present generally in the right ventricular wall and rarely in the left. Inflammatory cells prevailed in the degenerated areas.

Although the structure usually noted in cases of Fiedler's myocarditis has less degenerative change and rarely recorded areas of hemorrhage; nevertheless, since the entire scope of change in this heterologous group of conditions has not been finally determined, this case may be properly so designated. Certainly, pregnancy was not a factor and B avitaminosis was problematical as a factor.

Case No. 3: C. J., colored male, 39 years of age, entered Shreveport Charity Hospital February 20, 1949, was discharged March 3, 1949, and re-entered March 23, 1949, dying six days later on March 29, 1949. The chief complaints were shortness of breath, palpitation upon slight exertion and dependent edema. These complaints had become increasingly severe for the past two months. Recently there had been a frequent cough productive of white phlegm. He stated that at age 10 he had migratory joint pain without swelling or fever and continued to have some joint and muscle pain as an adult in cloudy weather.

On physical examination the blood pressure was 140/100 and the pulse rate 100. Moist rales were present in both lung bases. The heart was enlarged with the PMI in the sixth intercostal space well beyond the midclavicular line. There was a grade 3 systolic apical murmur, and also, on the second admission an apical diastolic murmur was heard. The liver was palpated two fingers below the costal border and was tender. There was 2 plus pitting edema of the lower extremities. The blood Kahn and Kolmer was 4 plus. The blood NPN was 71 mg. per cent and creatinine 2.6 mg. per cent. There was a leukocytosis of 32,000 with 67 per cent polymorphonuclears, 7 per cent lymphocytes and 6 per cent monocytes. An x-ray of the chest showed the heart enlarged 64 per cent above the calculated normal for his height and weight. The patient was treated with mercurial diuretics and digitalis and was discharged after twelve days edema free and with scant dyspnea. He was thought at this time to have rheumatic heart disease. Twenty days later he was readmitted complaining of extreme dyspnea, nausea, vomiting,

and pain in the upper quadrant of the abdomen. There was no edema present upon this admission. The remainder of the physical examination was similar to that of the previous admission.

Electrocardiograms on three occasions showed prolonged AV and intraventricular conduction, marked left axis deviation, and inversion of the T waves in Leads I and IV. In spite of diuretics, sedation and oxygen the patient remained dyspneic. Eleven hours before death he experienced severe upper abdominal pain, followed by profuse perspiration and extreme dyspnea. The pain and shocklike state persisted until his expiration, which occurred quietly after about eleven hours.

Autopsy showed a greatly enlarged heart with a rounded apex. The heart weighed 520 grams. There was hypertrophy of the papillary muscles and mural thrombi were present in both ventricles. No valvular or coronary vessel disease was seen. The myocardium was somewhat flabby and pale with many grayish white areas. There was a thrombus in a large branch of the pulmonary artery. There was chronic passive congestion of the liver and spleen, with infarcted areas in both kidneys. The femoral vessels showed no thrombi.

Microscopic study of the heart: Sections showed numerous focal areas of microbiosis of the myofibrils with fairly marked lymphocytic and monocytic infiltration. The myofibrils showed coagulation necrosis, staining poorly and showing fragmentation. The inflammatory cells appeared in clusters, but in some areas were scattered in the interstitial tissue of the myocardium. Another feature of the sections was diffuse scarring of the myocardium characterized by degeneration and fibrosis of the myofibrils.

These findings are characteristic of chronic interstitial myocarditis, and since no specific etiology of the lesion could be determined on clinical and histopathological examination, one is justified in making such a diagnosis.

Case No. 4: D. T., colored male, age 22, was admitted to Shreveport Charity Hospital on September 16, 1946, and expired the same day, after being in the hospital only six hours. The chief complaints were shortness of breath and swelling of the feet. The patient was semicomatose but history obtained from his wife indicated that he had had "rheumatism" for a number of years. About nine months before admission, his feet began to swell and he became progressively dyspneic. Four months prior to this admission, he was in the same hospital because of weakness, dyspnea, and pedal edema, and was diagnosed as having rheumatic heart disease. Severe symptoms had been present for three weeks prior to admission, principally pedal edema and marked dyspnea. On admission he was cold and clammy, with a blood pressure of 120/80. The heart was definitely enlarged to physical examination with both a systolic and diastolic murmur at the apex. There were no rales heard in the lungs. The liver extended three fingers below the costal border. Two plus pedal edema was present.

Postmortem examination showed the heart rounded and dilated with a transverse diameter in the chest of 19 cm.; the inside diameter of the chest being 27 cm. The heart weighed 560 grams. There were no vegetations and no stenosis of any valve. Microscopic study of sections from the heart showed hypertrophy, interstitial fibrosis, edema, considerable areas of scarring, and in the left auricle marked subendothelial fibrosis. There was no Aschoff nodules and no cellular inflammatory reaction, except in areas of scarring of considerable dimension where persivascular lymphocytic reaction prevails. Scarring was present around vessels. The pericardium was edematous and showed a few lymphocytes, but lacked the vascularization usually seen in rheumatic pericarditis. There was no valve deformity.

Cardiac enlargement of this proportion in the absence of definite deforming valve lesions or active rheumatic carditis, would render improbable the consideration of a rheumatic etiology for the chronic fibrotic changes present. The diagnosis of chronic Fiedler's myocarditis was therefore justified. The spleen, liver, lungs, and kidneys showed chronic passive congestion.

SUMMARY

We have presented 4 cases of isolated myocarditis, conforming in general to the clinical anl pathological picture known as Fiedler's myocarditis. The possible etiological factors have been discussed and the relationship to acute interstitial myocarditis seen with various infections emphasized. It is felt that Fiedler's myocarditis and the acute interstitial myocarditis seen with various infections are essentially the same, the chief difference being that the etiology of Fiedler's myocarditis is undetermined.

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DISCUSSION

Dr. Homer J. Dupuy (New Orleans): During routine chest x-ray studies on cases of virus pneumonia seen in the 64th General Hospital during the last war, six cases were found on my service to have an enlarged heart and abnormal electrocardiograms. With subsidence of the pneumonitis, x-ray studies revealed that the heart receded in size and the electrocardiograms reverted to normal. It was felt from the observations that the abnormal heart findings were probably due to a virus myocarditis. We also found several cases with abnormal electrocardiograms unassociated with enlarged hearts in cases of virus pneumonia. There were no appreciable alterations in the cardiac rate to account for the electrocardiagraphic changes. I think from these findings we can conclude that virus myocarditis can occur not infrequently as a complication of a virus infection elsewhere or may even occur as an isolated infection. Similar findings have been reported by other medical officers of the last war.

Dr. C. Thorpe Ray (New Orleans): The actual incidence of Fielder's myocarditis is unknown. It has been pointed out that the microscopic picture considered diagnostic of Fiedler's myocarditis may be limited to a small area of the heart. Many patients are seen with large hearts with persistent congestive failure in whom no etiologic diagnosis can be made. The length of life varies considerably and death may be sudden. In routine postmortem studies just a few sections are made and thus it is a matter of chance that the typical findings of Fiedler's myocarditis will come under the pathologisit's observation. A thorough search of multiple areas of the heart must be made. Only in that way will the true incidence of this disease be apparent.

MALINGERING

N. J. BENDER, M. D.
SHREVEPORT

When an individual consciously and deliberately imitates or exaggerates an illness for the purpose of attaining a particular gain, he is malingering. Without the element of frank dishonesty, the undue prolongation of an illness or the unwillingness to return to work, cannot be regarded as evidence of true malingering. The gain is more important to the sufferer than the discomfort entailed in maintaining the symptom. For example, a negro woman who had delivered her eighth child at Charity Hospital developed severe abdominal pain within an hour after the interne told her that she was to be discharged the following morning, which was her fifth postpartal day. The internes and visiting doctors then worked exhaustively in an attempt to make a diagnosis. Four days later the patient recovered and calmly told the psychiatrist that she was ready to go home. She confided that she had been allowed to remain in the hospital for nine days after the birth of her other seven babies and that after all the only "rest and waitin' on" she ever got was those nine days a year, following the birth of each child. This woman was malingering

Oberndorf in his book *Which Way Out* tells the story of a malingerer who successfully deceived the doctors and gained free winter lodgings. Jock MacKaye was a thin, hollow-cheeked man of forty who arrived at a New York State Hospital in the early fall. The commitment blank from Bellevue stated that he was hallucinating. The examiner at the hospital found Jock to be withdrawn, shy, and uncommunicative, but was unable to find evidence of hallucinations by indirect questioning. Finally,

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abandoning good interview technic, the doctor asked Jock bluntly, "Do you hear voices?" This was the only subject that Jock liked to discuss. He replied: "Voices talk to me all the time. The birds and the bees, they talk to me. They tell me to be a guid boy and Jesus will bless me . . . They're whispering right now, 'take guid care of yesself, be a guid laddie . . .'." Jock made an unusually good adaptation to the routine of the hospital; however, he never lost an opportunity to tell of his conversations with the birds and the bees. The hospital staff diagnosed Jock MacKaye as a schizophrenic. Early in April Jock began to tell his doctor that he could no longer hear voices. "Well, it's like this doctor, I starts to hear the voices in the autumn when the cold weather comes . . . they always stop in April. . . . Ye've got a grand hospital here. I spent last winter up in Massachusetts. Once I went up in Connecticut. . . . I save a wee bit of money this way, too. . . ." The hospital staff agreed that JockMacKaye was a clever malingerer.

The tendency to feign an illness is not limited to human beings. The opossum and and the fox feign death to turn away pursuing dogs. My son's wire haired terrier just this winter developed a counterfeit cough for the purpose of persuading his young master to let him continue to sleep in the house, a privilege that the dog had been granted when he had a severe bronchitis.

There are people, both young and old, who also control or try to control their environment with fake complaints. Mrs. Mary E., a woman sixty-six years old, deliberately falls or cuts her finger, whenever she feels neglected by her son. Incidentally, she plans to continue to get attention in this manner. Everyone is acquainted with the child who used the stomach-ache to avoid school. There is no end to the symptoms that people can use to control or to avoid a situation.

Although all malingerers are not "boarders" as were the negro woman and Jock, and although all malingerers do not resort to actual bodily damage as did Mrs. E., all malingerers are guilty of behavior which is planned for selfish gains. Since the behavior of the individual from the "normal" to the "psychotic" is purposeful, the malingerer's behavior, which is conscious, is often confused with that of the psychoneurotic, which is largely unconsciously determined. Conscious awareness of the intent in simulating or exaggerating an illness is not necessary in order that such behavior be purposeful. For example, the symptoms in hysteria are to some extent a solution to a problem without conscious recognition on the patient's part. The symptoms afford a certain relief from the anxiety or inner tension involved in a perturbing situation. While the behavior in both the malingerer and the hysteric is usually protective or wish-fulfilling in nature, the hysteric honestly believes that he is afflicted with the condition to which he attributes his alleged disability. The malingerer deliberately lies and adopts his behavior to gain some clearly defined external objective of which he is thoroughly aware.

Dunbar in her interesting book *Mind and Body* suggests that the fashion of the day has much to do with the selection of the "beloved symptom" in cases of malingering and hysteria. The great Prime Minister Gladstone is accused of inventing the "diplomatic cold" in order to avoid embarrassing political meetings. Elizabeth Barret Browning, a psychoneurotic, injured her spine when she was fifteen, but found illness such an acceptable means of escaping at least a part of her harsh father's tyranny that she developed "consumption", a popular disease for Victorian girls. Robert Browning rescued Elizabeth from both her bed and her environment. Since she no longer needed her symptom, she completely recovered. Allergy has served some of its victims well as a respectable affliction, but the virus seems to be gaining in popularity.

Litigation is an important determining factor in the patient's exaggerating or simulating illness and in perpetuating disability. These patients must be carefully studied in order not to confuse them with the psychoneurotic. Many malingerers seek compensa-

tion and dishonestly declare that they are suffering unendurable pain from even minor accidents. Backaches and headaches are among the most common complaints. In most cases the physician can reach a conclusion only after he has made a thorough study of the clinical picture in conjunction with a complete personality investigation, which will usually reveal that the patient has never been able to adjust to ordinary life situations. For example, Mr. M. filed suit for compensation for injury received when a short board fell across his head. This was Mr. M's fourth compensation suit over a period of ten years. He resisted physical examination. He answered all questions with caution, sarcasm, and rudeness. When asked how he was sleeping, he answered with a sneer: "In a bed, how do you think?" In this man's work record there were long periods of idleness for reasons that he could not explain. This man was a malingerer, but there were many psychoneurotic traits in his personality. It is indeed difficult always to make a correct differential diagnosis.

In evaluating the clinical picture of a patient, the physician must take into consideration the great variability of moral responsibility in different individuals. Many malingerers fall within a group classified as constitutional psychopathic inferiors. Such was the diagnosis of nineteen year old J. M. who had filed suit for compensation because of back pain that he attributed to a blow which he received while working as a roughneck on an oil well. Thorough and repeated examinations by an internist and by an orthopedic surgeon over a period of four months failed to reveal definite objective injury. The orthopedic surgeon noted that J. M. on several occasions bent over to tug and pull on his cowboy boots without showing any evidence of back pain, although during examination he had complained of excruciating pain on movement of his back to the slightest degree in any direction. It was also discovered that J. M. had a juvenile court record. His parents reluctantly revealed that their son had always been a problem to them; he was "selfish and headstrong". At ten years of age he began slipping out of his room to spend most of the night downtown. His mother stated that from early childhood he would tell a lie even when the truth would have served his purpose better. After punishment he would appear apologetic and remorseful, but would straight-way continue to do as he pleased, regardless of others. He had made good grades in school until truancy began in high school. At thirteen years, with another boy, he broke into a gasoline station. At fourteen years of age he served a period in the reformatory for car theft. At sixteen he disappeared from home and no one heard from him for two months. In short, the psychiatric evaluation of this boy revealed a personality which had always shown a lack of the ability to appreciate the nature and consequences of his acts; a life-long history of pathological lying; and a superficial, emotional reaction with little or no capacity for considering the feelings of others, or for comprehending moral responsibility.

Although definite criteria for detecting malingerers are impossible, there are some common factors. The malingerers tend to exaggerate their manufactured symptoms by constantly bringing them to the attention of the examiner. They lack sincerity. Sometimes their complaints are vague, but usually definite symptoms are chosen. They are cautious and seldom look the doctor straight in the eye; however, they no not lose an opportunity to steal a glance to see what impression they are making. A detailed history taken initially in the malingerer's own words is often contradicted later in spite of caution. Discrepancies appear frequently. Often the malingerer will forget to maintain his symptom when his interest is distracted by irrelevant conversation or when he is dressing or undressing. While the malingerer is greatly concerned about his symptoms, the hysteric often impresses one as being too little concerned. In addition, psychoneurotic reactions are usually sudden in onset, generally occur soon after an emotional shock, and are profound.

The diagnosis of a psychoneurosis is strengthened by evidence of an infantile personality as well as by motor and sensory hysterical conversion symptoms which the patient is not able to control. Those patients who further distort the presence of actual organic disease by psychogenic elaboration (either consciously or unconsciously) are the most difficult cases to diagnose. Masserman states that from the psychodynamic point of view "all neurotics tend toward conscious malingering as part of their total adaptations and all malingerers in their personality reactions are necessarily neurotic".

SUMMARY

A malingerer deliberately imitates or exaggerates an illness for the purpose of attaining a definite gain.

The psychoneurotic's behavior is also purposeful and directed toward a goal, but it is an unconscious phenomenon.

There are all degrees of conscious awareness between frank malingering and true psychoneurosis.

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THE PREVENTION OF DEAFNESS*

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The prevention of deafness is much too broad a subject to cover adequately in a brief discussion of this type, but a general survey of the problem and an attempt to emphasize a few methods of prevention will be made. Deafness as defined by the Committee on Definitions and Standards of Nomenclature of the Conference of Executives of American Schools for the Deaf[1] and approved by the American Otological Society[2] means a loss of the sense of hearing to an extent that hearing is nonfunctional for the ordinary purposes of life. In order to prevent deafness, hearing losses should be recognized early so for our purpose any hearing loss will be considered as a degree of deafness. Pain, vertigo, tinnitus, or any other abnormal sensation connected with the ear, may be the beginning of a condition which may go on to deafness.

ETIOLOGY

It is axiomatic that if the cause for a condition can be removed the development of a defect will be prevented. In deafness, we may go a step further in that removal of etiological factors may lead to a restoration of the auditory function. The causes of deafness are heredity, trauma, and diseases and abnormalties of the auditory mechanism.

Heredity is an important factor in the etiology of some types of deafness. Marriage of two deaf persons should be discouraged, especially if they have similar lesions, as the likelihood of their progeny having hearing defects would be greatly increased. Statistics show that with two otosclerotic parents nearly all daughters have otosclerosis, while only half to two-thirds of the sons have the condition.[3] Congenital familial deafness and Meniere's syndrome are also passed down to future generations.[4] Other types of congenital deafness are those due to congenital syphilis and to intra-uterine rubella (German measles). The avoidance of contact with rubella by pregnant women, especially in the first third of pregnancy, and the purposeful exposure of young girls to this usually mild disease for the establishment of immunity are steps that may be taken to avoid occurrence of congenital defects.

Trauma is another factor entering into the production of hearing defects. Acoustic trauma is perhaps more important in civil

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life than concussion or penetrating wounds, because of frequent exposure to noise in industry and in aviation. Deafness produced by noise is usually in the upper part of the conversational range and higher (2000-6000) and tends to be reversible, but prolonged exposure leads to permanent defects. For this reason every effort should be made to reduce noise exposure and to protect ears by the use of ear defenders.

Injury from blast may rupture the tympanic membrane which may be followed by infections in the middle ear. Similarly, a dive or fall into water may injure by the sudden concussion. Concussion sometimes produces injury to the cochlea or the central connections of the auditory mechanism. Efforts should then be made to protect the head and the ears in particular from concussion injury.

DISEASES AND ABNORMALITIES

Any condition which obstructs the external ear canal will result in interference in the reception of sound by the air conduction route. Inflammatory swelling and foreign matter in the canal are transitory conditions which do not warrant comment here, but congenital atresia of the canal and new growths are conditions the correction of which will result in restoration of hearing if the rest of the mechanism is normal.

Disease of the internal ear and nerve deafness present a challenge in the prophylaxis of deafness. Labyrinthitis when it occurs usually leaves hearing defective, so conditions that may lead to it should be promptly cared for. Infections of the ear and mastoid should be adequately treated, and infection in the teeth, tonsils, and sinuses, should be eradicated. Syphilis may lead to involvement of the internal ear. Labyrinthine hydrops (Meniere's Disease) warrants serious medical treatment if hearing is to be preserved. Furstenberg's[5] salt free diet with ammonium chloride or potassium chloride and Horton's[6] histamine treatment are most used. Toxic damage by quinine and streptomycin should be avoided. The dosage of 1.0 gm. streptomycin daily in divided portions, as used now, rarely affects the hearing. Mumps, measles, and meningitis may cause toxic nerve deafness.

Otosclerosis has been mentioned in connection with inherited forms of deafness. Much of our knowledge of the etiology of this abnormal formation of spongy bone in the otic capsule is theoretical. However, the observation that the onset of symptoms frequently occurs at the time of stress or emotional strain suggests that suspected otosclerotics should avoid unnecessary stress.

OTITIS MEDIA

The field in which our effort to preserve hearing and prevent deafness has been most successful is in the care of conditions involving the middle ear. We may have acute or chronic conditions. Acute purulent otitis media may be due to pyogenic organisms or to the acute infectious diseases (especially measles and scarlet fever). Early incision of the drum and drainage of middle ear abscesses shortens the course of the infection, and with the help of sulfa, penicillin, and other antibiotic drugs the ear is usually returned rapidly to normal and hearing loss is avoided.

Acute catarrhal otitis media or serous otitis media is the result of blocking of the eustachian tube.[7] When the tube does not open normally to allow entrance of air into the middle ear there is an absorption of air followed by transudation of serum into the cavity. This is prone to happen when there is acute inflammation in the nasopharynx, nose, or sinuses, and it is often precipitated by a change in altitude with resultant change of atmospheric pressure and inability of the subject to equalize the pressure in the middle ear with atmospheric pressure (aero-otitis). Proper treatment of the inflammatory condition in the nose and throat followed later by gentle inflation of the eustachian tube is often sufficient to return the ear to normal, but in more persistent cases it may be necessary to do a tympanotomy to drain the serous fluid from the middle ear. (Figure 1.) Infection may take place in catarrhal otitis;

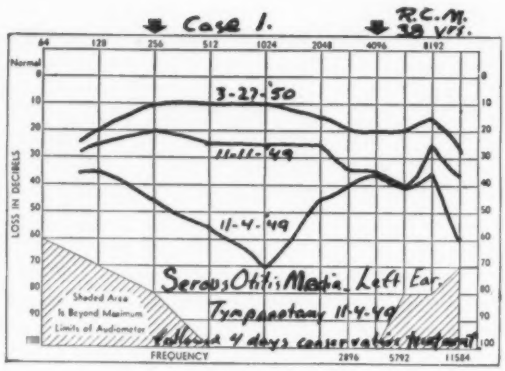

Figure 1

then a purulent otitis media exists. Predisposing factors should be removed in order to prevent recurrences.

Chronic otitis media may be purulent, catarrhal, or due to one of the granulomatous diseases (syphilis, tuberculosis). The purulent form is the continuation of an acute purulent otitis media which has failed to get well for one reason or another. The ear may not have been cleaned and drained adequately or mastoiditis may exist. The eustachian tube may not have been patent or nasopharyngeal infection may be present.

Chronic catarrhal otitis media, as the name implies, is a persistent type of otitis in which there is eustachian tube obstruction with decreased pressure in the middle ear and retraction of the tympanic membrane. There may or may not be fluid in the middle ear, but it is sterile.

Syphilis and tuberculosis of the middle ear were in time gone by the cause of deafness, but with adequate antisyphilitic therapy in the former and streptomycin in the latter, the outlook is considerably more favorable. Prompt diagnosis is essential.

Catarrhal otitis media and purulent otitis media have several points in common. An obstruction of the eustachian tube is a prime factor in etiology and must be removed to effect a cure. The hearing tests are similar with a negative Rinne test, Weber test lateralizing to the most involved ear, probably but not always a greater loss in low tones than in high, and rarely a loss of more than 40 to 45 decibels by air conduction. Salvage of these ears is best accomplished in children and young adults. However, our efforts are often rewarded by success even in the aged. Time is an important factor—the longer a hearing defect has existed, the less likely it is that hearing will be improved.

Eustachian tube obstruction (acute or chronic) may be due to abnormal lymphoid tissue in or about the pharyngeal orifice of the tube, to adhesions, inflammation in the nose or pharynx, or to structural abnormalities. Lymphoid tissue in the form of enlarged adenoids or even smaller follicles in or near the tube may be responsible for blocking, or perhaps after adenoidectomy regrowth or adhesions may prevent the passage of air through to the middle ear. Acute obstruction is usually the result of an inflammatory process occurring in the nose, sinuses, pharynx, or tonsils of an individual already partly blocked. Obstruction will be favored by bony or cartilaginous abnormalities in the nose or nasopharynx such as deviation of the nasal septum, atresia of the posterior nares, and other developmental defects. These should be corrected if possible when tubal obstruction exists.

The treatment of hyperplastic lymphoid tissue in the nasopharynx has been much discussed of late. There is little difference of opinion in the method of handling a large mass. It should be removed surgically (figure 2) and if obstruction persists it

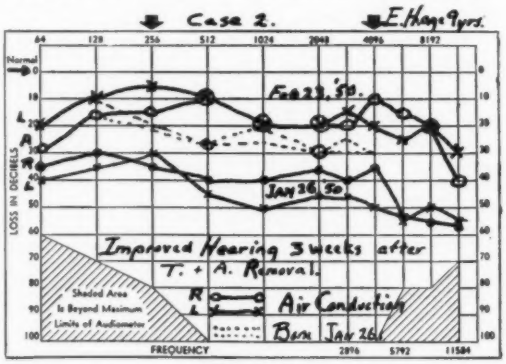

Figure 2

may be necessary to use radiation (figure 3). In small masses, diagnosed by posterior

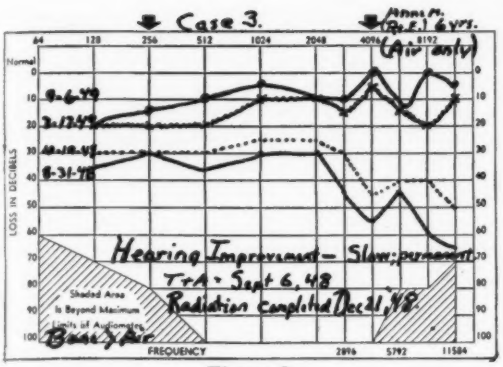
Case 3.
Hearing Improvement – Slow; permanent
Tr A - Sept 6, 48
Radiation completed Dec 21, 48
Shaded Area is Beyond Maximum Limits of Audometer
FREQUENCY
LOSS IN DECIBELS

Figure 3

rhinoscopy or nasopharyngoscopy, radiation is the method of choice either by roentgen ray or preferably by the use of the nasopharyngeal radium applicator pioneered by Crowe and Burnam of Johns Hopkins.[8, 9] (Figure 4.)

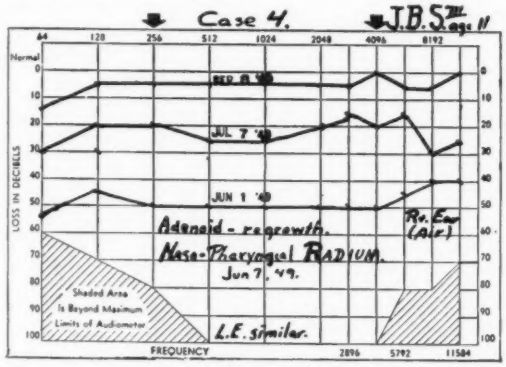

Figure 4

The method used at present is that of Crowe. The patient lies on a comfortable low treatment table and with or without topical analgesia the radium applicator is placed so that the monel metal capsule containing 50 mg. of radium salts is just medial to the pharyngeal orifice of the eustachian tube. The radium stays in place twelve minutes, then the procedure is repeated on the other side (or two applicators may be used simultaneously). Three such treatments are given at intervals of two weeks and no more should be given for at least a year.

Some question has been raised as to the safety of using radiation of the nasopharynx,[10] but Crowe in an editorial note in the Yearbook for 1949 states that since 1924 he has treated several thousand patients without harm to anyone or to himself. Recent literature contains many reports that agree with Crowe.[11-14] My cases date back more than ten years without ill effects and many show dramatic results.

CONCLUSIONS

1. Many cases of deafness are preventable if they are recognized and properly treated.

2. Removal of etiological factors in many will prevent deafness or restore hearing.

3. The return to normal of the eustachian tube is one of the greatest factors in the prevention of hearing loss.

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DISCUSSION

Dr. Paul Azar (Lafayette): What is your opinion about early tonsillectomy? Many doctors in small towns get little children, 1 to 5 years of age, with repeated attacks of tonsillitis. These doctors are asked to remove the tonsils, yet there is so much lymphoid hyperplasia after removal. What age is the time for tonsillectomy?

Dr. Garland Walls (New Orleans): There is no age limit for tonsillectomies. Bad tonsils and adenoids should come out. Do not take them out

unless there is justification for it. I have taken them out at 14 months and have seen them removed at 6 months. One of those removed at 14 months had had nine middle ear abscesses and any measure was justified to stop that, and so it did.

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STREPTOMYCIN THERAPY FOR CHILDHOOD TULAREMIA

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JACQUES D. WILKINSON, M. D.
LONGVIEW, TEXAS

The advent of the antibiotic era has afforded the pediatrician numerous effective therapeutic weapons against the infectious diseases, but in none have the results been more striking than in the use of streptomycin against tularemia. Following Heilman's initial report[1] concerning efficacy in experimental infection, there appeared a number of studies[2] concerning clinical application in the various types of human tularemia. However, the majority of these reports have dealt with adult therapy. Minden and Springer[3] successfully treated an 11 year old boy with oculoglandular tularemia, and Cawley and Lafsky[4] have described the typical ulceroglandular type seen in a 7 year old boy, also cured by streptomycin.

Increased interest in tularemia as a disease of major concern to the pediatrician, especially in this area, is well demonstrated by the recent report of a ten year survey in Arkansas,[5] in which 110 cases of a total of 704 were listed as occuring in the preschool and school child, ranking these groups just below the agricultural worker and housewife in frequency of infection.

At Shreveport Charity Hospital, there were seen during the period from May 1943, through July 1949, a total of 48 cases of tularemia in childhood. A critical survey of the clinical aspects of this study has been published previously.[6] This report will deal specifically with the 24 cases which have received streptomycin therapy since this antibiotic became available late in 1946.

The data presented in Table I gives the essential information concerning each of the children so managed. Inasmuch as the available supply of streptomycin was markedly limited at the onset, and there was no criterion of dosage other than the reports of adult infections, there was considerable variation in the therapeutic courses, especially for the early cases. The average total dosage administered was 8.37 grams, given for an average duration of 7.46 days; during the latter portion of the series, dosage averaged one gram daily for five to six days, using 125 mg. at three hour intervals. Even this dosage is in excess of that recommended by Foshay,[7] who suggests 0.5 gram daily for two days followed by 0.25 gram daily for four days, or, as an alternate course, 0.5 gram daily for six days.

The remarkable clinical response can be best demonstrated by illustrative case reports:

CASE REPORTS

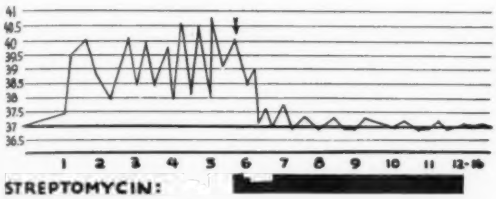
L. K., a six year old negro boy, was admitted to the hospital on May 20, 1947, with his three year old sister, having a history of headache, loss of appetite, and listlessness for the previous two days. His sister had similar but more severe symptoms, entering in a semicomatose condition. The uncertain history, given by a relative afforded no evidence of animal contact or tick bite. Physical examination revealed a lethargic colored male who showed little of note other than generalized hyperactive reflexes with marked increase in hamstring tonus, slight tonsillar enlargement without exudate, and small cervical and inguinal lymph nodes. The abdomen was soft and nontender, with no enlarged organs and no tenderness or rigidity. Temperature on admission was 37.5 C. Penicillin was begun with a dosage of 50,000 units initially, followed by 30,000 units every three hours. However, the temperature rapidly rose to 39.5, then 40, and began to follow a septic type of picture. Agglutination reactions showed positive for Typhoid "O" in dilution of 1:40, *Brucella abortus* 1:20, Proteus X 19 1:320, and tularemia negative. Examination of the cerebrospinal fluid revealed no significant data. On the third hospital day (sixth day of disease) the clinical and agglutination pictures were essentially unchanged, and cultures of the blood, stool, and urine were negative. The patient's sister expired on the following day; autopsy findings

From the Pediatric Service, Shreveport Charity Hospital.

Read before the Seventieth Annual Meeting of the Louisiana State Medical Society, Baton Rouge, La., April 26, 1950.

showed the presence of extensive tularemic infection. On that same day the patient became lethargic and appeared to be seriously ill. Streptomycin was given in an initial dosage of 200 mg. followed by 100 mg. every three hours for eight doses, then 100 mg. every four hours for six doses. There was an immediate, dramatic response (Figure 1), with

CASE 21



AGGLUTINATIONS:

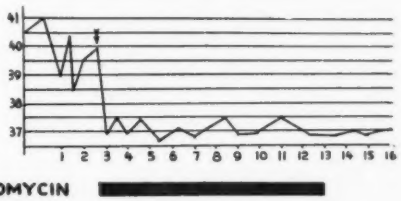
Ty-O-	1:40	1:40	1:40	
B.ab.-	1:20	1:20	1:40	
Pr.OX19-	1:320+	1:320	1:320	
Tul.		Neg.	1:40	1:640

Figure 1

return of the temperature to normal levels. As more of the agent became available, the streptomycin dosage was increased to 200 mg. every three hours for five days, with complete recovery following a total of 4.6 grams. The tularemia agglutination became positive (1:40) on the eighth day, and by the eleventh day had risen to 1:640. The history was then obtained from a relative concerning ingestion of rabbits and squirrels at frequent intervals. This, together with the autopsy findings in the sibling, caused us to classify this case as one of ingestional typhoidal tularemia, successfully treated with streptomycin.

E. L. McG., a three year old colored girl, was admitted on October 9, 1947, with a history of high fever of four days' duration, sore throat, and swelling in the neck. She had been treated at home for "acute tonsillitis" without appreciable improvement. The patient's mother stated that she had removed a tick from the child's neck several days prior to the onset of symptoms. Physical examination revealed an acutely ill colored girl with a temperature of 40.5 C., hyperemic pharynx, large tonsils with a shaggy necrotic exudate, and enlarged left postauricular and cervical lymph glands. With persistence of a severely septic course after thirty-six hours of penicillin therapy, the child was given a therapeutic trial with streptomycin, using a dosage of 125 mg. every three hours. There was an immediate response (Figure 2) with rapid return of temperature to normal and general clinical improvement. Agglutination reactions on the sixth day of illness were reported positive in dilutions of 1:80 for Typhoid "O" and *Brucella abortus*, and 1:160 for tularemia. By the eleventh hospital day (fifteenth day of disease)

CASE 27



AGGLUTINATIONS:

Ty.O.	1:80		
Tul.	1:160	1:1280	1:2560
HETEROPHILE		1:224	1:896

Figure 2

the tularemia agglutination had risen to 1:1280, with the heterophile antibody reaction reported two days later as 1:224. These two agglutinations later rose to 1:2560 and 1:896 respectively. Streptomycin was continued for ten days, giving a total dosage of 10 grams.

DISCUSSION

A multiplicity of medicinal agents, ranging from arsenic to sulfanilamide[8] were tested against tularemia in the past, but none achieved any measure of success prior to the development of specific antiserum by Foshay.[9] This remained the treatment of choice until the appearance of streptomycin, which has now been proved superior to both the immune and hyperimmune sera.[10] The excellent results obtained in the 2 cases discussed above were duplicated in each of the 22 other children treated, even though septicemic involvement occurred in 10 cases. Recent experimental studies have indicated that aureomycin[11] may be of value in tularemia therapy, especially in the adult. We have as yet had no experience with this newer antibiotic against this infection, but feel that streptomycin will in all probability remain the treatment of choice for the critically ill child.

In spite of the relatively large doses used, there were no instances of toxicity or sensitivity noted; with the lower dosage and shorter course now advocated, this danger will be lessened even more. Institution of therapy early in the course of the disease appeared to afford no alteration to development of natural resistance, as in every case there was elevation of the agglutination

TABLE 1
STREPTOMYCIN THERAPY FOR CHILDHOOD TULAREMIA

Age Race Sex	Type of Infection	Day Begun	Amount and Interval	Total Dosage in Grams	Duration in Days	Clinical Response
6 C. M.	Pharyngotonsillar	15	250 mg. stat; then 125 mg. q. 3 h.	6.125	6	Afebrile in 24 hrs.
4 C. M.	Ulceroglandular septic	8	300 mg. stat; then 125 mg. q. 3. h.	8.3	8	Afebrile in 72 hrs.
* 6 C. M.	Typhoidal	6	200 mg. stat; then 100 mg x 14; 200 mg. q 3. h.	4.6	7	Afebrile in 48 hrs.
5 W. F.	Pharyngotonsillar septic	8	400 mg. stat; then 200 mg. q. 3. h.	14.6	9	Afebrile in 48 hrs.
** 3 C. F.	Pharyngotonsillar	6	125 mg. q. 3. h.	10	10	Afebrile in 12 hrs.
10 C. M.	Ulceroglandular	14	125 mg. q. 3. h.	4	4	Afebrile in 12 hrs.
5 C. F.	Ulceroglandular septic	30	150 mg. q. 3. h.	8.4	7	Afebrile in 12 hrs.
4. C. F.	Pharyngotonsillar septic	8	125 mg. q. 3. h. x 16 200 mg. q. 3. h. x 21	15.2	9	Afebrile after 24 hrs. of larger dose
5 C. M.	Pharyngotonsillar septic	8	as above	as above	as above	Afebrile after 48 hrs. of larger dose
6 C. M.	Ulceroglandular	17	150 mg. q. 3. h.	7.2	5	Afebrile in 48 hrs.
8 C. M.	Ulceroglandular	10	125 mg. q. 3. h.	5	5	Afebrile in 48 hrs.
11 C. M.	Ulceroglandular	12	125 mg. q. 3. h.	5	5	Afebrile in 48 hrs.
11 C. M.	Ulceroglandular	12	150 mg. q. 3. h.	12	10	Afebrile in 72 hrs.
5 C. F.	Glandular	15	200 mg. q. 3. h. x 24 200 mg. q. 6. h. x 16	8	7	Afebrile on admission No change in nodes
8 C. M.	Ulceroglandular septic	12 35	150 mg. q. 3. h. x 64 125 mg. q. 3. h. x 32	9.6 4	8 4	Questionable relapse Good response each time
4 C. M.	Ulceroglandular septic	5	125 mg. q. 3. h.	6	6	Afebrile in 96 hrs.
10 C. M.	Ulceroglandular	23	125 mg. q. 3. h. x 32 50 mg. q. 3. h. x 64	7.6	12	Afebrile in 48 hrs.
8 C. F.	Ulceroglandular	17	150 mg. q. 3. h.	10	8	Afebrile on admission
12 C. M.	Glandular	14	125 mg. q. 3. h.	9	9	Afebrile in 48 hrs.
11 C. F.	Glandular	27	125 mg. q. 3. h.	5	5	Afebrile in 48 hrs.
5 C. M.	Typhoidal	6	125 mg. q. 3. h.	5	5	Afebrile in 36 hrs.
11 C. M.	Typhoidal	6	125 mg. q. 3. h.	5	5	Afebrile in 24 hrs.
11 C. M.	Glandular	16	125 mg. q. 3. h.	6	6	Afebrile on admission
9 C. M.	Ulceroglandular	8	125 mg. q. 3. h.	10	10	Afebrile in 12 hrs.

*L. K. (Case 21)—see Figue 1
**E. L. McG. (Case 27)—see Figure 2

titer regardless of the interval involved in the assumption of treatment.

There have been two deaths from tularemia on the Pediatric Service since the advent of streptomycin, as unfortunately, neither of these children received the benefit of this agent. One was a 14 months old colored boy who expired within six hours of admission to the hospital, with autopsy findings of extensive tularemic involvement of the entire intestinal tract; the other was the sister of the first case discussed previously, inadvertantly diagnosed as "meningitis versus rickettsial disease". With the disclosure of the true nature of her infection, we were able to successfully apply streptomycin therapy for her sibling, with the gratifying results already mentioned.

The rapid and often dramatic response to streptomycin, encountered in every child so treated in this series, testifies to the remarkable efficacy of this antibiotic against the *Pasteurella tularense*. Since the diagnosis of tularemia in childhood may be extremely difficult, especially in those cases in which adequate history is lacking, one should feel justified in including a therapeutic trial with streptomycin for every severely ill child who fails to respond to other treatment. The laboratory confirmation of tularemia appears too late in the course of the disease to warrant delay, and a twenty-four or forty-eight hour course of streptomycin may well afford the difference between diagnosis in the test tube or on the autopsy table.

SUMMARY

1. Twenty-four cases of tularemia in childhood have been successfully treated with streptomycin.

2. Administration of 1 gram daily for five to six days, given as 125 mg. doses at three hour intervals, proved adequate in most cases.

3. Any critically ill child in whom the diagnosis is questionable, and who shows failure to respond to other treatment, should receive a therapeutic trial with streptomycin, as the results may prove lifesaving.

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STUDIES OF THE DIETS OF PREGNANT WOMEN IN MISSISSIPPI: II DIET PATTERNS*

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AND
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JACKSON, MISSISSIPPI

INTRODUCTION

The purpose of this section of the studies is to report what pregnant women in a low socio-economic group in the rural South are eating. The findings are those of a dietary survey conducted from February 1948, through May 1949, in eleven counties in southern Mississippi. Four hundred and two women participated. Of these, 372 were colored and 30, white. The patients were being furnished prenatal care by the county health departments of the Mississippi State Board of Health.

An accurate inventory of the food habits of the patients was desired for the information and use of the obstetric consultant, the nutritionists, and the local health department staffs. To know what these women were eating was requisite in the preparation of our instructional diet leaflets, particularly those directed against excessive weight gain and toxemia.[1] To go too far afield from their already established habits promised very little cooperation. Spot checks had given the impression that the

*The authors gratefully acknowledge the assistance of Dr. Roy E. Butler, Senior Surgeon, USPHS.

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diet was very poor. Studies done elsewhere have suggested a correlation between poor diet and our unimpressive eclampsia, stillbirth, prematurity, neonatal, and maternal death rates. With the results of a careful study, the need for more nutritional education could be gauged and a program directed. It would be a basis for nutrition teaching in the clinic and in the school room. This survey is amenable to repetition in a number of years to measure the progress that will be made. In the past quarter of a century the quality of the diet in this group of patients has undoubtedly improved, but there is a dearth of old reports that can be held as measuring sticks against present day habits.

The nutritional status of a group can be assayed by a combined evaluation of the diet pattern, physical condition, and biochemical studies. This is an attempt to supply the diet pattern.

The educational level in this group is the lowest in the state. The majority live in rude homes that deserve to be called cabins. No electricity or plumbing is the rule. For the greater part, cooking is done on a wood stove. These women are predominantly rural and agricultural. A fraction live on the fringes of towns of over 20,000 population but their entire manner of living is similar to the completely rural participants. From this same source of patients, one author found that 52.5 per cent of 1669 pregnant women had hemoglobin levels below 11 grams/100 ml.[2]

THE RECORD COLLECTING

The diet history used here was a one-day food record taken by personal interview. The patient was put at ease by a chatty conversation and the twenty-four hours preceding the hour of interview covered. The meetings were scattered about equally in the forenoon and the afternoon. Over 300 histories were taken by one author (A.G.K.) and the remainder by two other Public Health Nutritionists. The one-day record seemed the most suitable for our purposes. It was recent enough to be accurate; these people could not be depended upon to recall what they had eaten over a more remote period. The keeping of a written record was impracticable because so many were illiterate. An unexpected interview allows no opportunity for modification to please the reporter. The number of patients for whom the diet was distorted by missed meals due to distance from home to health department was not significant. Other merits of the one-day dietary record have been annotated.[3]

We believe that these diet records portray what the women are actually eating. The nurses, who discuss diet at each prenatal visit, vouch for them as representative. They have made home visits and in many instances have had contact with the patient and family extending over a number of years. If there was any chance for inaccurate reporting it was in the direction of making the intake better than it actually was to please the interviewer (who wanted the patient to eat a better diet). A check on the accuracy was the monotonous similarity in the diet in each county for each season.

The patients had had previous contact with Board of Health personnel genuinely interested in their habits and offering welcomed advice. The nutritionists had a broad knowledge of the class of people they were questioning. They were familiar with their speech, habits, and philosophy. They knew how to establish rapport and get accurate answers. Results from any other type of interviewer would have been nearly worthless.

The participants in this sampling had no notice that their diet was to be scrutinized. They simply dropped in for a prenatal visit. There was no selection in the form of appointments made for particular patients because a nutritionist was to be in attendance. The patients were in all months of pregnancy but because of their tendency to defer seeking antepartum care until late in pregnancy, a majority were in the second semester. The anorexia of the first trimester had no appreciable influence on these records.

Many of these women had a recent improvement in their food habits, resulting

from previous prenatal visits at which a good diet was outlined and emphasized by the nurses. Those who had prenatal care in previous pregnancies had a knowledge of protecting foods superior to others in their stratum. Spontaneous remarks illustrating the effects of previous contacts were (1) "The nurse told me that I ought to drink milk but I don't like it." (2) "I get my red meat every day. The nurse said I needed it." (3) "My sister gave me a paper (nutrition leaflet) like that." (4) "I drink my quart of milk every day."

This record of food habits is naturally not representative of the State of Mississipi as a whole but does show what a sizable group of women bearing children in the state are eating. Of the deliveries in Mississippi, 37 per cent are attended by midwives. The group of women studied here are typical of those women who engage midwives; indeed over 90 per cent were thus attended.

The interviews are fairly equally scattered throughout the months of the year although clinic attendance is somewhat influenced by season. Attendance is lower during rainy periods in the winter and when the crops require attention.

An attempt will be made in the following sections of these studies to correlate diet and maternal health.

SOME REGIONAL PECULIARITIES

The interrogators were struck by the comparative absence of food likes and dislikes among the patients as compared to their own inclinations. The patients were directed mainly by availability and habit. On the Gulf Coast many husbands work in the fish industry and sea food is found on the menu once or twice a week, occasionally four or five times. Patients on the best diets were the ones who by their own statement had husbands who were making "good" salaries. The authors, in Part I, have recorded the remarkable custom of including clay (25 per cent) and laundry starch (39 per cent) in the diet.[4]

Cooking methods are primitive. They have had little opportunity to learn to prepare food better in domestic science classes or by imitation. Each cooks the way her mother did. They have no recognition of the fact that there are ways to prepare food that are better than others. They are familiar with the word "vitamins" but not with the preservation of them in the kitchen. Some illustrating statements are (1) "I cook greens 'til they fall to pieces." (2) "I put the greens on early in the morning and cook 'em 'til dinner." (3) "I cook 'em 'till they're tender." (4) "I set the greens on the back of the stove for supper." (5) "I fries nearly everything."

Seasonal variations usually can be found in the diet. Some seasons and their favorites are: spring and early summer, turnip greens; later summer, collards and cabbage; fall and winter, field peas; December and January, occasionally rabbit; winter and spring, red beans. An unpublished survey of the diets in northern Mississippi by one author (A.G.K.) showed a more marked seasonal variation because the natives raised a greater proportion of their food and bought less in the stores. In the north, a larger percentage were tenant farmers. As a result of these home gardens the iron content in the north was good and poor in the south. In the south, greater amounts of starch in the form of rice, grits, and bread are consumed.

This class of people had a tendency not to sit down as a family to meals three times a day but to eat irregularly all day. Sixty-four per cent ate sporadically, with either less than three meals a day, or had no scheduled meal time. The last meal of the day was any time between 3 and 9 o'clock. The choice of collards or turnip greens with cornbread for the first meal of the day was a deviation from the usual American pattern. When greens were eaten early in the morning they had been cooked the day before. Two pertinent quotations were (1) "I don't eat at any certain time. I just eat all day long." (2) "I never set down to eat. I just eats when I'm hungry."

An impediment to improved nutrition has been certain shopping difficulties in rural southern areas. The inaccessibility of large markets forces the inhabitants to

buy at "cross-roads stores" where variety is limited and prices high. A visit to four "cross-roads stores" revealed that there was a total of five varieties of animal protein offered: canned tripe, sardines, mackerel, sausage in oil and corned beef. Only two fresh vegetables were for sale, onions and Irish potatoes.

COMPOSITION OF DIETS

When our data were analyzed it was found that only 6 per cent of the 402 women were getting the National Research Council's recommended allowance of specific nutrients for pregnancy (Table 1). Calculation of the nutrients was based on Bowes and Church's tables.[5]

TABLE 1
RECOMMENDED DAILY NUTRITION ALLOWANCES OF NORMAL WOMEN IN PREGNANCY. FOOD AND NUTRITION BOARD, NATIONAL RESEARCH COUNCIL

Calories	Proteins	Calcium	Iron	Ascorbic Acid
2400	85 grams	1.5 grams	15 mgs	100 mgs

No attempt was made to compare the diets of the whites and non-whites because the distribution by race did not permit statistical appraisal. The observation was made that all of this fortunate 6 per cent were non-whites.

TABLE 2
DIET SURVEY OF 402 MATERNITY PATIENTS IN SOUTHERN MISSISSIPPI

	Total	Percentages
No. having no breakfast	116	29
No. having no lunch	48	12
No. having no supper	91	23
No. having no milk	184	46
No. having 1 glass milk	100	25
No. having 2 glasses milk or more	73	18
No. having 3 glasses milk	45	11
No. with no lean meat (not salt meat)	157	39
No. with 1 or more servings of lean meat	245	61
No. with no eggs	229	57
No. with no other proteins	361	90
No. with 1 or more serving of other proteins (dried peas or beans)	65	16
No. with no vegetables or fruits	47	12
No. with 1 serving of fruits or vegetables	131	33
No. with 2 servings of fruits or vegetables	111	27
No. with 3 servings of fruits or vegetables	62	15
No. with 4 servings of fruits or vegetables	51	13

Table 2 summarizes some of the interesting findings in the diet content and meal patterns. Outstanding facts were the number who omitted entire meals. A 46 per cent for those who had no milk was a disappointing figure. That one-third was found to have had only one serving of fruit or vegetables seems an important finding.

Foods that often appear on the menu are: spaghetti, grits, rice, bread, salt pork (fried or boiled). Some foods that appear seldom are: cheese, milk, raw vegetables, and fruits. Eighty-nine per cent did not receive the recommended one quart of milk. The amount of candy eaten was insignificant. Twenty per cent had a soft drink in the twenty-four hours before interview.

Examples of the worst menus are:

No. 1

Breakfast
3 tablespoons grits
1 tablespoon butter
2 pieces toast
1 cup coffee
Dinner
1 candy bar
1 apple
1 soft drink
No supper

No. 2

No breakfast
Dinner
1 root beer
2 plates field peas
4 biscuits (large)
Supper
½ plate water gravy
1½ plates fried okra
2 biscuits (large)

Menus selected as typical were:

No. 3

Breakfast
1 egg
1 piece salt pork
1 tablespoon syrup
2 large biscuits
Dinner
2 pork chops
1½ plates field peas
1½ plates corn
4 pieces cornbread
1 piece coconut cake
No supper

No. 4

Breakfast
3 tablespoons grits
1 fried egg
1 piece salt pork
2 pieces toast
Dinner
1 plate stewed beef
1 plate turnip greens
2 pieces cornbread
Supper
2 pieces fried fish
2 pieces cornbread

Menus considered above average were:

No. 5

Breakfast
3 tablespoons grits
1 fried egg
1 tablespoon butter
2 pieces toast
2 glasses milk
Dinner
1 piece fried steak
1 plate collards
1 helping candied sweet potatoes
½ plate rice
2 pieces cornbread
2 glasses lemonade
Supper
1 peanut butter sandwich
1 fish sandwich
2 glasses buttermilk

No. 6

Breakfast
1 large dish of oatmeal
1 glass milk
1 rounding teaspoon sugar
1 egg
1 biscuit
Dinner
½ plate of butter beans
1 piece steak
½ plate fried okra
½ plate corn
2 glasses buttermilk (large)
2 pieces bread
Supper
1 plate field peas
1 tomato
1 plate okra
1 helping fried potatoes
2 pieces cornbread

In taking these records it became apparent that if a patient were able to obtain only occasionally a certain type of food she had an unusually large intake of it for a brief period. This certainly was influenced by the lack of refrigeration. Pay day was an immediate prerequisite. One patient had hamburger, three pieces of fried pork, four pieces of fish and two pieces of liver in one day. Another had four eggs, and one woman had two pieces of steak, one pork chop, and two eggs. Still another had five oranges and a large glass of orange juice.

IRON

Only 6 per cent were receiving the recommended allowance of iron. The intakes ranged from 3 milligrams to 27 milligrams with a median of 11.2. The principal sources of iron were meat, turnip greens, and collards.

VITAMINS

We did not feel that the record was accurate enough to quote the median intake on several vitamins. The food was not weighed and the methods of preparation created uncertainties. Vitamin C was generally decimated in cooking. As the providers of vitamin C were citrus fruits and raw vegetables, and other sources were inconsequential, we had an accurate gauge of the vitamin C intake (Table 3).

TABLE 3
VITAMIN C INTAKE BASED ON THE INTAKE OF ONE OR MORE SERVINGS OF CITRUS FRUIT OR RAW VEGETABLES

Poor	72%	(No citrus fruits and no raw vegetables)
Fair	19%	(One serving of citrus fruit or raw vegetables)
Good	9%	(Two or more servings of citrus fruit or raw vegetables)

CALORIES

The principal sources of calories and carbohydrates were bread, grits, spaghetti, rice, fried foods, and salt pork. Other sources were sweet potatoes and syrup. In a sample of 100 diets the intake ranged from 600 to 4,000 calories with a median of 2,600.

CALCIUM

As the only considerable sources were milk, collards, and other greens, the intake of calcium could be determined (Table 4). Only 3 per cent had enough milk and green vegetables daily to meet the recommended allowance.

TABLE 4
CALCIUM BASED ON 0-4 CUPS OF MILK DAILY PLUS ONE OR MORE SERVINGS OF GREEN VEGETABLES

Poor	68%	(One cup of milk* daily or less, plus no green vegetables)
Fair	29%	(One to two cups of milk* daily, plus one serving of green vegetables)
Good	3%	(Three to four cups of milk* daily, plus one or more servings of green vegetables)

*36% of the milk was in the form of buttermilk and 2% was clabbered milk.

PROTEINS

The principal sources of protein were lean meat, eggs, milk, and field peas. Sixty-one per cent of the protein eaten by these patients was in the form of meat or eggs. Sixteen per cent had no protein of animal origin or insignificant portions. The meats that appeared most often on the menu were pork chops, ham, steak, roast, fish (always fried), crab (in coastal counties), hamburger, luncheon meat, potted meat, and sardines. Cheese was an exception on the menu; it had been eaten by only 5 of the 402 women. Eighty-nine per cent had inadequate milk intake, which would have been a substantial source of protein. Fifty-seven per cent had not had the one egg we advised. The protein intake for the entire group is listed in Table 5.

TABLE 5
PROTEIN INTAKE OF 402 WOMEN

Poor	61%	(Below 60 grams per day)
Fair	33%	(60 to 84 grams per day)
Good	6%	(85 or more grams per day)

In 402 diets the daily protein intake ranged from 11 grams to 140 grams with a median of 60 grams.

PHYSICAL STATUS

Sometime during the pregnancy each woman had a physical examination by the local health officer. No case of frank avitaminosis was uncovered. This is not surprising in view of the increasing mass of evidence of the lack of correlation between apparently deficient diets and the supposedly classical signs of specific vitamin deficiency.[6] This study seems to add strength to the proposition that the minimal requirements are much less than the Recommended Nutrition Allowances (Table 1). Unfortunately, exact technics for finding subclinical deficiencies were not available. The only biochemical study on hand from this group is the previously mentioned high rate of anemia. Serious vitamin deficiency disease has become much less frequent in Mississippi. In 1915 there were 1535 deaths from pellagra and 30 in 1948.

When the list of the weights on first visit of 349 of these women is inspected, 31 per cent are found to be over 150 pounds and 23 patients over 200 pounds (Table 6). No inconsistency appears to exist between our record of calories consumed and these weights. Butler[7] has emphasized obesity as a form of malnutrition, the result of eating too much of the wrong type of food.

TABLE 6
MATERNITY PATIENTS' WEIGHTS UPON FIRST VISIT TO CLINIC

	Number	Percentages
100 pounds and less	9	3
101 to 150 pounds	231	66
151 to 200 pounds	86	25
Over 200 pounds	23	6
	349	100

SUMMARY

The dietary of 402 pregnant women in a typical southern rural area is reviewed. The technic of the interview is described. The women are from a low socio-economic group. The diet is monotonously poor and determined largely by poverty and ignorance. Only 6 per cent are receiving the National Research Council's recommended allowance of specific nutrients for pregnancy. The number of calories seems to be the only standard of a satisfactory diet that is met. Iron, vitamin C, calcium, and protein intakes are unsatisfactory by the usual standards. One-third have only one serving of fruit or vegetables in a day. Eighty-nine per cent are not receiving a quart of milk a day. Definite evidence of vitamin deficiency is not found in these women. Some sample diets are provided.

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NOTES ON MEDICAL HISTORY OF THE PANAMA CANAL

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NEW ORLEANS

INTRODUCTION

The author is fully aware that much has been said and written about the medical conquests on the Isthmus of Panama. However, after having served four years as a commissioned officer of the U. S. Navy, two of these years having been spent in and around the locale of these achievements, the author began to feel a more keen appreciation of the seemingly insurmountable obstacles which were met and overcome by these courageous individuals, some of whom are discussed below. This is the origin of my interest in the subject.

Every available opportunity was seized upon to see, at first hand, the medical installations now present on the Isthmus, and to gather information from some of the "Old Timers" now living there who were present in those turbulent days of the actual construction of the Canal. This paper is derived partly from information gained there, plus references to the literature on the subject. References are listed at the conclusion of this paper.

It is only possible to allude to the color, tragedy, pathos, and ultimate success of sanitation and medicine in the Canal Zone as it unfolded on the stage of history at the turn of the twentieth century. Present visitors to the Isthmus probably find it hard to conceive that such a healthful area was, less than fifty years ago, a tempestuous, disease-ridden place visited only by the adventurous and/or curious, and even then with many misgivings on their part.

The triumph of man against yellow fever and malaria in Panama occupies one of the brightest pages in the history of preventive medicine. What had formerly been a disease-infested land was miraculously transformed in the short time of little more than one year from the "white man's grave" into a veritable garden spot of the world by the practical application of academically proved health safeguards. Many men are responsible for the healthful conditions of the Panama Canal Zone but the chief organizational genius and *tour de force* behind this success story is William Crawford Gorgas, M. D. One author flowingly describes him as "America's Ambassador of Mercy, the benefactor of humanity, and the redeemer of the tropical world".

EARLY ERA

For centuries, European nations had sought to find shorter routes to the Far East for their sailing ships, but in vain. So far as we can now ascertain, Columbus was the first white man to visit Panama, and on his third expedition to the New World (1498), he spent some time on the northern (Atlantic) side of Panama, at Bocas del Toro, Limon Bay, Porto Bello, and Belen. The era of the *conquistadores* followed soon thereafter, and they became especially active in Panama when the fortunes of the fabulous Inca Empire became known in the Old World. Gold and silver were transported, overland, from the Southern (Pacific) borders of Panama to be loaded at Porto Bello for trans-shipment to Spain. This prompted naming the land "Castilla del Oro". Meanwhile, diseases were exacting a tremendous toll, especially among the white population, causing this land to be known abroad as the unhealthiest region of the Americas.

MODERN ERA

After years of turbulent history, including the romantic days of the Spanish Main buccaneers, it may be said that the modern era began for Panama in 1880 when the French undertook to construct a canal

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across the Isthmus of Panama, connecting the Atlantic and Pacific Oceans, and capable of carrying ocean-going vessels. They organized *La Compagnie du Canal Interoceanique de Panamæ*. They had just returned from their recent successes with the Suez Canal and, under the leadership of their inspired "Conqueror of Suez", Ferdinand de Lesseps, they began their gigantic construction project.[15] The Medical Department of the French Canal Company was very active and took measures to provide what would have been adequate medical care for the workers, under average tropical conditions. Their medical directors were aware, of course, of the appalling death rates from yellow fever and malaria, but ideas as to the etiology were extremely nebulous, to say the least. Fomites and miasma were then being mentioned as the more probable causes. The French chose the Pacific side of Panama as the General Headquarters of their medical operations and built, in 1882, their main hospital along the southern and eastern fronts of Ancon Mountain. It was appropriately named, Ancon Hospital. This hospital had a 700 bed capacity and was of the pavilion type. A modern dairy supplied fresh milk, and drinking water was gotten from chemically tested springs. The excreta disposal system was adequately maintained by a bucket system of closets. The nursing staff consisted of Catholic sisters, assisted by negro maids and orderlies. The medical staff was headed by a Dr. LaCrosade, and French physicians staffed his hospitals and dispensaries. Apparently, all was provided except *protection against Stegomyia fasciata, the mosquito carrier of infection* (which, as mentioned above, was unknown to the French at that time). Twelve hundred deaths, mostly among Frenchmen, occurred in Ancon Hospital alone. Dr. Porras, a former president of the Republic of Panama, recalling the days of his early youth, said, "I can still remember the impossibility of trying to sleep at night because of the constant and tormenting bites of the mosquitoes . . . tormentors so numerous that by clutching at the apparently empty air, I caught handful after handful. On every hand one encountered well beloved friends hastening home in the grip of malarial chills, or some equally pernicious fever".

The French Canal Company, which had existed nine years, ended in bankruptcy and the death of 22,189 laborers. In addition, when the United States took charge of the Canal in 1904, a yellow fever epidemic was sweeping away the inhabitants by the thousands. Such was the medical state of affairs when in 1904, Colonel Wm. C. Gorgas, MC, USA, was appointed Chief Sanitary officer of the Panama Canal Zone, under authority of the Isthmian Canal Zone. This post was organized by the President of the United States under the Spooner Act (of January, 1904).

Dr. Gorgas, son of a Confederate Army general (who was later president of the University of the South, Sewanee, Tennessee), was well primed for his enormous task in Panama. He had recently succeeded in ridding Havana, Cuba, for all practical purposes, of yellow fever for the first time by 1901. This feat was accomplished by putting to practical use the theories of Dr. Carlos Finlay, a Cuban physician, and the later proof by Dr. Walter Reed and his assistants, Drs. Aristides Agramonte, Jesse W. Lazear, and James Carroll, that the mosquito *stegomyia fasciata* was responsible for the spread of yellow fever from person to person. Gorgas followed the same measures which were instituted in Cuba. The Isthmus was cleaned up and kept clean; modern sanitary laws were applied to the supply of drinking water and the sewage disposal, and finally a successful campaign was waged against mosquitoes. Yellow fever quickly disappeared, and the incidence of malaria was reduced more slowly but just as surely. One year after Gorgas took charge, yellow fever and malaria were wiped out on the Isthmus of Panama.

Mr. Wellcome, the founder of the Tropical Research Laboratories at Khartoum, visited Panama at the request of the U. S. Secretary of War in 1910 and reported, "I have never seen anything anywhere that equalled the perfection of organization, sys-

tematizing, and direction that I found under the administration of General Gorgas. Gorgas had gathered about him a wonderful staff". This reporter went on to state how "he was taken on long cruises through the reeking Panama swamps to see at first hand what had to be overcome. One of the various serious problems which has baffled many workers in the humid tropics has been how to effectually destroy, and keep under control, the rank plant life that springs up in a night with overwhelming rapidity at the water's edge and in marshy places, forming cup-like receptacles that are filled by the rain and make abundant breeding places for mosquitoes. The Panama Sanitary Staff succeeded in checking this growth by means of a powerful blast of burning oil sprayed on to the roots of the undergrowth, and they scattered sand over the cup-like holes made by crabs on the seashore, as mosquitoes can easily breed in salt water. The cost of Gorgas's operations was one cent a day per man. Fifty-sixty thousand men were employed in the Canal Zone and malaria was practically stamped out among them". Mr. Wellcome continued, "a finer piece of economic health control would seem to be impossible".

In 1914, ten years after Gorgas began his work, the death rate in the Canal Zone for 1,000 of population was six, as compared with 14.1 per 1000 in the U. S. A. During the ten years of construction by the United States, there were lost by death, 17 out of each 1,000 employees per year—i.e., from the whole force of 39,000 men, 663 died each year, and for the whole construction period there was a total of 6,630 deaths. The Ancon Hospital was completely refitted by Dr. Gorgas and converted into a modern hospital of 1500 beds. New equipment was brought in and all facilities begun by the French in this hospital were expanded manyfold. (The author can truthfully state, from many personal visits to this hospital, now named "Gorgas Hospital," that it is efficiently run and can compare favorably with U. S. hospitals of a similar type). Colon Hospital on the northern (Atlantic) side of Panama formed the other terminus of the medical line and thus there were two base hospitals. In between, there were divisions of the territory into as many medical districts as there were sanitary districts (twenty), and each of these districts had a small hospital of 20 to 100 beds, where treatment could be carried out where it would have been disadvantageous to transport the patient to either of the base hospitals. Many of the districts had several villages within their limits where the laborers lived. Each of these villages had in it a small hospital of 5 to 15 beds where the sick could be kept until moved to the district hospital. There were forty of these sub-district hospitals, and these were generally called "rest camps". This gave, all told, sixty hospitals. Screened hospital cars were run over the railroad between Colon and Ancon, morning and evening, in both directions. Each car was manned by a trained white male nurse and a negro assistant. Ambulances met the trains at each end of the line and carried the sick to the base hospitals. One or more dispensaries were maintained in each medical district also. Each dispensary was headed by a druggist with at least one assistant. The district physician presided over the district dispensary and advised without charge anyone who applied for treatment and furnished him with medicines if he was unable to pay.[8]

Dr. Gorgas completed his work at Panama and lived to achieve other successes before retiring from the Army. Among the personal rewards he received in recognition for his outstanding work are the Mary Kingsley Medal from the Liverpool School of Tropical Medicine (1907), and honorary degrees from the Universities of Pennsylvania, the South, Harvard, and Johns Hopkins. In addition, he was elected president of the American Medical Association in 1909-10. In 1914, Oxford University conferred on him an honorary D. Sc. degree, and the American Museum of Safety awarded him their Gold Medal. Also, in 1914, he was promoted to the office of Surgeon General of the U. S. Army. The following year he was promoted to the permanent rank of

major general. In 1915, he received a vote of thanks from Congress for his services in connection with construction of the Panama Canal.

Following his victories against disease at Panama, Dr. Gorgas undertook a series of investigations of an epidemic form of pneumonia among the miners of the Rand in South Africa for the British, and of yellow fever in South America for the Rockefeller Foundation. His last project was undertaken at the urgent request of the British to serve on a yellow fever commission in West Africa. He never reached Africa for death resulted from cerebral hemorrhage on July 4, 1920, while he was in London making preparations for his newest venture. He was given a British state funeral with full military honors in St. Paul's Cathedral, London, following which his body was brought back to this country in a British warship. The Gorgas Memorial Laboratory was erected in Panama in honor of this great man, and, from several personal visits, the author can report that this fitting memorial is worthy of him.

Today, the health of the Canal Zone populace is remarkably good. The health plans carried through by Gorgas have been expanded tremendously. The Chief Health Officer of the Canal Zone, who is a medical officer of the U. S. Army, has charge of all medical and allied activities on the Isthmus. He makes an annual "Report of the Health Department of the Panama Canal", published by the Panama Canal Press, Mount Hope, C. Z. and the author suggests it as a complete source of information on the morbidity and mortality rates from diseases and injuries, together with other vital statistics relating to the population of the Canal Zone and the cities of Panama and Colon.

A brief resume of vital statistics in the Canal Zone is shown in Tables 1 to 5.

TABLE 1
DEATH RATE PER 1,000 POPULATION—ALL CAUSES

	Calendar Year				
	1947	1946	1945	1944	1943
Canal Zone	6.0	6.31	5.12	6.13	6.24
Panama City	7.96	9.44	9.42	10.57	10.49
Colon	9.47	12.00	12.32	11.55	12.11

TABLE 2
NUMBER OF DEATHS AND ANNUAL RATE PER 1,000 POPULATION—CALENDAR YEAR 1947

	Canal Zone	
	Number	Rate per 1,000
Organic diseases of the heart	33	0.69
Cancer of various organs	41	.86
Pneumonia	10	.21
Diseases of the arteries	32	.67
Tuberculosis	12	.25
Apoplexy	15	.31
Nephritis (acute and chronic)	19	.40

TABLE 3
BIRTH RATE PER 1,000 POPULATION

Canal Zone	Calendar Year				
	1947	1946	1945	1944	1943
White	55.92	44.69	32.23	32.65	27.21
Colored	19.17	18.92	18.47	19.07	15.25

TABLE 4
DEATH RATES OF INFANTS UNDER ONE YEAR OF AGE PER 1,000 LIVE BIRTHS

Canal Zone	Calendar Year				
	1947	1946	1945	1944	1943
White	14	17	24	17	24
Colored	45	63	44	43	51

TABLE 5
MALARIA

Calendar Year	Rate per 1,000
1943	15
1944	13
1945	13
1946	12
1947	12

(There was one death from malaria among employees during the calendar year 1947).

Although the figures shown in the charts are rather remarkable, it should be stated that the death rate for the Canal Zone is artificially low in comparison with Panama City and Colon because of:

1. The selective residential group in the Canal Zone;

2. Ex-employees generally leave the Isthmus after retirement.

The birth rate has only limited significance (Table 3) due to methods currently in vogue upon which the rates are calculated.

SUMMARY

A brief resume of the medical history of the Panama Canal, including *La Compagnie du Canal Interoceanique de Panama*, and

the United States Panama Canal Company, is given. A chronological development of medical progress, beginning with conditions of Panama in the days of the *Conquistadores,* through the present day, and ending in the eradication, for all practical purposes, of malaria and yellow fever from the Isthmus of Panama is discussed. The medical safeguards of the French, followed by the successful tactics of Gorgas are described at length. Mention of the state of health as now found on the Canal Zone completes this report. The words on the Grand Seal of the Canal Zone Isthmus of Panama, "The Land Divided, The World United", are more true today than ever before but only as a result of the Franco-American medical axis of LaCrosade—Gorgas, and an expansion of the latter's viewpoints on tropical preventive medicine.

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(Most of the information in tables numbered 1 through 5 was obtained from References (1), and (2) above).

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THE TOXIC EFFECTS OF ASPIRIN

Aspirin, or acetylsalicylic acid, has been in use for half a century, and is probably the most widely used drug in the world today. It appeared at a time when the use of opium had been freer than it should have been and almost indiscriminate in some respects.

Aspirin fills the need for the relief of many physical pains and mental conditions. In certain instances it is superior to other remedies. Because of such widespread use, it has been accepted by many that there is an almost universal tolerance for the drug, and conversely, that poisoning is not to be feared.

During the first 25 years since its introduction in 1889, aside from the statements in textbooks that the toxic effects resemble those of salicylic acid, there were comparatively few comments in the literature as to possible ill effects that might be produced by aspirin. In recent years, many articles have appeared and they call attention to its untoward effects; these are disturbing and may be alarming.

Aspirin was introduced to provide a drug of its desirable properties, which would also avoid the uncomfortable effects of salicylic acid and the salicylates. In many respects, this was the case; however, in certain individuals taking large or small doses, toxic symptoms may appear. In some there is a relationship between the dose and the symptoms, and in others profound disturbances follow administration of small doses. These symptoms are similar to those of salicylic acid poison and may include tinnitus, deafness, headache, nausea, vomiting, hyperpnea, profuse sweating, cyanosis, acute pulmonary edema and circulatory collapse, semi-stupor and possibly coma. In the more chronic cases there may be gastric pain, burning, pyrosis, abdominal pain, restlessness, constipation and apathy or irritability. In some instances of acute or chronic types, there may be fever, erythematous rash and sleeplessness. An additional group of symptoms which may come from the toxic effects are those connected with hemorrhage. There may occur epistaxis, hematemesis, melena and hematuria. Some articles have appeared showing that increased hemorrhage after tonsillectomy may be produced by aspirin, locally applied or taken internally. In this connection, Link, Huebner and others, studying dicumarol, have made clarifying observations as to how these may occur. They showed that dicumarol may be qualitatively reduced to salicylic acid, and that salicylic acid caused a decrease in the prothrombin level in rats which had been kept on a diet already low in vitamin K. However, others have shown that when vitamin K is fed with the salicylic acid, there will not be a fall of prothrombin. This anticoagulant potency of aspirin has been

utilized in the treatment of coronary thrombosis by Gibson.

Hartmann has found that acute salicylate poisoning in infants and young children may produce a bicarbonate deficit acidosis and an acid urine, in which condition 50% may die. This disturbance of the acid-base balance has been found by others. In one case, a non-specific toxic encephalopathy was reported by Ryder et al. Instances of acute circulatory collapse are known in which cyanosis and pulmonary edema produced the appearance of acute congestive failure. Another alarming effect has been that of choking and respiratory distress producing a condition resembling asthma. One patient, when more than 40 grains of aspirin was taken in one day, was subject to the appearance of nausea, headache, fever up to 100°, and an erythematous rash. Others have recorded instances of severe hematemesis or melena. Relief of these untoward effects, as would be expected, comes when the drug has been withdrawn. In the acute phases in children, Hartmann advises infusions containing sodium lactate or sodium bicarbonate. The acute deficiencies of the circulation and the asthma syndrome in adults have been met by the injection of adrenalin.

Gastric irritation from aspirin has recently been more widely recognized than formerly. Various combinations of alkali with aspirin have become available in order to minimize this feature. These combinations have relieved the stomach discomforts ordinarily caused by aspirin in many patients.

Recognition of aspirin poisoning has been difficult. The drug may intensify the symptoms it was taken to relieve. The ill effects may appear just after its ingestion or they may develop after weeks of what seems to be satisfactory tolerance for the drug. Accordingly, identification of aspirin as the offender will depend upon the physician's being alert to any of the symptoms of salicylism which may occur during its administration.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

AMA MEETING—JUNE, 1950

Following is partial report of the recent meeting of the American Medical Association held in San Francisco, as submitted by Dr. George F. Lull, Secretary and General Manager of the Association:

"With a total registration of 23,655, the American Medical Association closed a very successful meeting in San Francisco recently. The figure includes 10,119 fellows and members.

"It was the biggest attendance of any A. M. A. session ever held in San Francisco. The A. M. A. met there five times during the last 35 years. The first A. M. A. session in San Francisco, held in 1915, drew 2,307 physicians while the last meeting—held in 1946—had an attendance of 7,655.

"Dr. John W. Cline, San Francisco, assistant clinical professor of surgery at Stanford University Medical School, and a very active member of the A. M. A. Campaign Coordinating Committee, is the new president-elect of the A. M. A.

"Dr. Cline, who has had a notable career in medicine since his graduation from Harvard University in 1925, delivered a powerful acceptance speech before the House of Delegates after he was notified of his election.

" 'We are fighting for the freedom and the medical welfare of the people of our country,' he said. 'Our cause is estimable. Our position is strong.

" 'Our major dereliction has been the failure to develop to its full potential the ag-

gregate strength of the individual members of the profession. I do not refer to dissident individuals or rump organizations of left wing orientation. These are numerically and intellectually insignificant and do not merit our attention.

" 'Most of our state associations and county societies have responded well. A considerable majority of our members have provided splendid support but far too many have not. This condition arises, not from a difference of principle, but from failure to understand the problem.

" 'The active and vigorous participation of this group is essential and must be obtained at the local level.'

"Among other officers elected by the House of Delegates was Dr. R. B. Robins, Camden, Ark., as vice-president. Officers re-elected were Dr. J. J. Moore, Chicago, treasurer; Dr. F. F. Borzell, Philadelphia, Speaker of the House; Dr. James R. Reuling, Bayside, N. Y., Vice Speaker, and Dr. George F. Lull, secretary.

"Dr. Leonard W. Larson of Bismarck, N. D., and Dr. Thomas P. Murdock, of Meriden, Conn., were elected to the Board of Trustees.

"The House of Delegates had a busy session, acting on more than 170 pieces of business.

"The House adopted the controversial and much-discussed Hess report, which is aimed at taking hospitals out of the practice of medicine. My office is now at work preparing a comprehensive analysis of the action of the House in this matter. When completed, it will be published in the A. M. A. Journal, and given wide publicity through the press. Some time will be required to make such an analysis because of the necessity of studying the stenotype report and comparing it with the Reference Committee report.

"The House also adopted the Board of Trustees' resolution advocating the Association's continued opposition to Reorganization Plan No. 27.

"The Reference Committee on Legislation and Public Relations urged adoption of a resolution which opposed subsidization of medical education.

" 'On the basis of the broad general principle established by the Supreme Court in 1942 that the government may regulate that which it subsidizes, the Committee believes that any bill providing federal subsidization for medical education will be detrimental to medical education', the Reference Committee report said, adding: 'It is the opinion of the Committee that the A. M. A. should continue to oppose legislation subsidizing medical education until and unless a method can be designed which would make ultimate federal control of our medical schools impossible.' "

In a subsequent report Dr. Lull advised that the House of Delegates of the A. M. A. also set A. M. A. dues for the ensuing year at $25.00 and that subscription to The Journal of the A. M. A. will be included in the dues. Fellowship in the Scientific Assembly was preserved and Fellowship dues were set by the Board of Trustees at $2.00 per year. Fellows may elect to take a special journal in lieu of The Journal of the A. M. A., which they would ordinarily receive as members, Dr. Lull stated, however, this privilege is not extended to members who are not Fellows of the Scientific Assembly.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

NAVY ASKS INACTIVE RESERVE, MEDICAL DENTAL AND NURSE CORPS OFFICERS TO VOLUNTEER FOR ACTIVE DUTY

Due to the shortage of physicians, dentists and nurses to meet the present day obligations, the Surgeon General of the Navy, Rear Admiral Clifford A. Swanson, asks that these members of the inactive Naval Reserve volunteer immediately for extended active duty. Medical and dental officers in the ranks of Commander and below and nurses in the ranks of Lieutenant and below will be accepted.

The recently authorized increase in the overall manpower strength of the Navy requires the services of additional Medical Department personnel to serve in the continental United States and overseas.

Members of the above reserve components volunteering for active duty should apply to the Chief of Naval Personnel, Navy Department, Washington 25, D. C.

SOUTHERN MEDICAL ASSOCIATION ST. LOUIS MEETING

The forty-fourth annual meeting of the Southern Medical Association to be held in St. Louis, November 13-16, 1950, will be highlighted by one of the most complete programs ever presented at any medical meeting. On the first two days, Monday and Tuesday, there will be presented four general clinical sessions covering the whole field of medicine, each session followed by a question and answer period. Some of the most outstanding men of medicine will appear on this program, presenting the latest in technics and therapeutics. Wednesday and Thursday will be given to section meetings. Thirty-two sessions will be held covering every phase of medicine. Section officers are busy securing the best possible speakers for these sessions. This St. Louis meeting will truly be the outstanding medical meeting of the year designed especially to aid physicians in the South in their daily practice.

Scientific exhibits, wherein the latest work in research in all phases of medicine are visually and graphically presented, will further add to the completeness of the meeting.

As has been the case at all Southern Medical Association meetings, the technical exhibits, sometimes called commercial, will consist of exhibits presented only by outstanding, ethical business firms. Only those products that are needed by physicians in their daily practice of medicine will be on display.

Hotel reservations will be handled through a Housing Bureau set up by the local committee. The Bureau will give those who wish reservations the hotel of their choice so long as that hotel has available the kind and price of accommodations desired. In writing for hotel reservations one should name the hotel of his choice, giving a first, second and third choice, indicating kind and price of accommodations desired, expected date and time of arrival, and names of all persons who will occupy accommodations. Address HOUSING BUREAU, Southern Medical Association, 911 Locust Street, Room 406, St. Louis 1, Missouri.

General Headquarters will be the Kiel Municipal Auditorium where all meetings and scientific and and technical exhibits will be held.

FELLOWSHIPS IN STUDY OF ARTHRITIS

The Arthritis and Rheumatism Foundation is offering fellowships for research in the basic sciences related to the study of arthritis. These fellowships carry a stipend of from $4,000 to $6,000, depending upon the needs and ability of the worker, and run for a period of one year. The fellowships will begin in July 1951, although earlier appointments will be considered by the committee.

The Foundation is anxious to back a candidate, rather than a project, an institution, or a hospital. It hopes to arouse interest in arthritis in a wider circle of medical investigators and to encourage able, inquiring minds.

Applications should be sent to the Arthritis and Rheumatism Foundation, 535 Fifth Avenue, New York 17, New York by January 1, 1951. Notification of the fellowships granted will be made March 1, 1951.

If any applications are received by September 15, 1950, they will be acted upon at that time and notification made immediately.

FELLOWSHIPS IN CARDIOLOGY

The Louisiana Heart Asociation announces two fellowships in cardiology, to be known as the Charles Payne Fenner, Jr. Memorial Fellowship and the Leon H. Rittenberg Fellowship; duration of one year, grant $2,400.00 to $3,000.00 each. These will be open to doctors having previously served at least one year of internship and one or more years of formal resident training in internal medicine or pediatrics. The purpose is to further research and provide additional clinical training in cardiovascular disease. Applications should be mailed to Dr. Allan M. Goldman, Secretary-Treasurer, Louisiana Heart Association, 3439 Prytania Street, New Orleans, La.

UROLOGY AWARD

The American Urological Association offers an annual award of $1000.00 (first prize of $500.00, second prize $300.00 and third prize $200.00) for essays on the result of some clinical or laboratory research in urology. Competition shall be limited to urologists who have been in such specific practice for not more than five years and to men in training to become urologists.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Palmer House, Chicago, Illinois, May 21-24, 1951.

For full particulars write the Secretary, Dr. Charles H. de T. Shivers, Boardwalk National Arcade Building, Atlantic City, New Jersey. Essays must be in his hands before February 10, 1951.

FEDERAL MEDICAL POSITIONS OPEN

Medical Officer positions in the Federal Service paying $5400, $6400, and $7600 per year will be filled from an examination recently announced by the Director, Eighth U. S. Civil Service Region, Saint Paul, Minnesota. Positions paying the above cited salaries are now vacant and there is an immediate need to fill these positions. Medical doctors who have just completed their internship are eligible for the positions paying $5400. Medical doctors with one or two years experience performing responsible medical doctor duties are eligible for the positions paying $6400 and $7600 per year respectively.

Applications for these positions will be accepted until further notice. Application forms may be obtained at any first or second-class post office. A copy of the examination announcement may be obtained by writing the Director, Eighth U. S. Civil Service Region, Saint Paul Post Office and Customhouse Building, Saint Paul 1, Minnesota.

WOMAN'S AUXILIARY OF THE LOUISIANA STATE MEDICAL SOCIETY

STATE LEADER IS ELECTED BY AMA WOMEN

For the first time a Louisianian has been named president of the Woman's Auxiliary to the American Medical Association—Mrs. A. A. Herold of Shreveport, founder of the Woman's Auxiliary of the Louisiana Medical Society in 1929.

A past president of the Caddo parish auxiliary, Mrs. Herold served the state group as its first vice-president, later becoming president. She is past president of the Southern medical auxiliary, and has held numerous state and national offices and chairmanships.

A leader in community affairs, the Auxiliary's new leader has been chairman of the home front committee, office of civilian defense; chairman of Red Cross surgical dressing groups in her parish; board member of the Shreveport Community Chest and of the Hephzibah Home for Unmarried Mothers; Caddo parish commander of the American Cancer Society Field Army 1; board member of the Shreveport Council of Social Agencies; past president of the Department Club, and past vice-president of the Shreveport branch of the American Association of University Women.

She succeeds Mrs. David Allman of Atlantic City, N. J., as the Auxiliary's national leader, Mrs. Daniel Murphy, New Orleans, publicity chairman of the State Auxiliary, announced.

ORLEANS PARISH

New Orleans is indeed very proud of the honor which has come to the city and the state through the essay contest sponsored by the Auxiliary this spring. The high school student who placed second in the local contest won fourth prize in the National Contest sponsored by the American Association of Physicians and Surgeons. Miss Rose N. Agnew of Metairie High School is the young lady who has brought this honor to the South for the first time in the four years the contest has been offered to high school students.

Public recognition was given to this highly important honor by one of the very prominent civic groups of the city—the Chamber of Commerce at one of their weekly luncheons, which was attended by several hundred members of the Chamber. The Acting President of the Chamber of Commerce, in very appropriate ceremonies, presented Miss Agnew with the prize money—a $25 check from the American Association of Physicians and Surgeons. Mrs. Clara Agnew, mother of the winner, and Mrs. Edwin R. Guidry, contest chairman and several other Auxiliary members were guests of Dr. Edwin L. Zander on this occasion. Dr. Zander had the honor of presenting the awards to the local winners of the contest.

BOOK REVIEWS

Thrombosis in Arteriosclerosis of the Lower Extremities: By Edward A. Edwards, M. D., F. A. C. S. Springfield, Charles C. Thomas, 1950. Pp. 74. Price $2.00.

Edwards' book on thrombosis in arteriosclerosis of the lower extremities is a monograph in the American Lectures in Circulation, which are under the joint editorship of Irvine H. Page and A. C. Corcoran. It describes in considerable detail and with great lucidity the role thrombosis plays in the precipitation of ischemic catastrophes in the arteriosclerotic extremity. The material dealing with etiology, pathology, clinical picture, and treatment is well organized and well presented. The discussions, in fact, are remarkably complete, in spite of the small space which they occupy. More than half of the book is occupied with a series of case reports chosen, as the author notes in his preface, to illustrate particularly the complications of the disorder, the difficulties of treatment, and the correlation of the clinical and pathologic pictures. The preponderance of cases coming to amputation or autopsy, as he points out, might seem to exaggerate the possibility of an unhappy outcome, but one must agree with him that there is full reason to emphasize the potential seriousness of thrombosis in arteriosclerosis of the lower extremities. One must also agree with him that immediate lumbar sympathectomy is the most significant single item of treatment available today. One week later the same operation is done on the contralateral side if the indication exists. The bilateral procedure is practical in young, vigorous persons.

The format of the book, like that of other Thomas publications, leaves nothing to be desired. The American Lecture Series texts, convenient in size, modest in price, and excellent in content are filling a useful place in medical literature. This particular text is a worthy addition to the series and is heartily recommended to both internists and surgeons.

FREDERICK FITZHERBERT BOYCE, M.D.

Clinical Pathology, Application and Interpretation: By Benjamin B. Wells, M. D., Ph.D., Philadelphia, W. B. Saunders Co., 1950. Illus. Pp. 397. Price $6.00.

The author is primarily interested in helping physicians use the clinical pathology laboratory to its best advantage. The book gives recommendations as to laboratory procedures and the interpretation of results. Each subject is introduced with a brief discussion of the theory and clinical manifestation, followed by the presentation of the most useful laboratory aid. Technics are not discussed. Numerous tables are presented enabling the reader to get information quickly. Figures are few and most of these have been borrowed. References to the literature are very sparse; others on disputable subjects should be included such as O. P. Jones' summary of the characteristics of the megaloblasts in *Archives of Pathology*, 1943. A few things are mentioned too briefly, notably the acid phosphatase in carcinoma of the prostate.

PHILIP PIZZOLATO, M.D.

Quinidine in Disorders of the Heart: By Harry Gold, M. D., New York, Paul B. Hoeber, 1950. Pp. 115. Price $2.00.

A most thorough and yet brief discussion of quinidine by one of the eminent authorities in cardiology. Written for the use of the general practitioner, it can be read with profit by the specialist. The subject is definitely presented on the Gold standard.

I. L. ROBBINS, M.D.

For the New Mother: By Mildred V. Hardcastle, R. N. Philadelphia, John C. Winston, 1948. Illus. Pp. 159. Price $2.00.

This is just another of the many books written for the lay public these days. It reminds me of my feeling for a new vitamin preparation—just another preparation for the same purpose. It does explain the self demand schedule and meets the many problems of the first year in a friendly easily understandable manner.

SUZANNE SCHAEFER, M.D.

Chemical Developments in Thyroidology: By William T. Salter, M. D., Springfield, Charles C. Thomas, 1950. Charts. Pp. 87. Price $2.00.

This fascinating little book was written by the Professor of Pharmacology at Yale, and it astounds one more conversant in clinical problems with the tremendous advances being made in the studies on thyroid cellular physiology and pharmacology.

The discussion pertains mainly to goiterogenic substances and their capability to "block" thyroid activity and the studies on physiology of the thyroid with radioactive iodine and its projected use in hyperthyroidism and malignancy.

Iodine-bound serum protein exerts a metabolic effect similar to thyroxine; and diiodotyrosine and thyroxine are formed in other tissues of the body besides the thyroid though in less quantity. "There is ultimate possibility that chemical hyperthyroidism might be treated with an analogue of thyroxine which could block the effect of hormone on general body tissues. Possibly also such analogues might 'deceive' the pituitary and so suppress thyrotropic secretion." These and many other such problems are discussed in the light of recent advances.

The reviewer believes that all clinicians and surgeons and biochemists who teach students aspects of thyroid disease would benefit by reading this book.

HOWARD MAHORNER, M.D.

Progress in Clinical Endocrinology: Edited by Samuel Soskin, M. D., New York, Grune & Stratton, 1950. Illus. Pp. 641. Price $10.00.

This is a series of brief reviews written by many of the outstanding authorities in the field of clinical endocrinology covering all aspects of this very broad subject. As should be expected from a book which attempts to cover so much, the reviews are in general brief but excellent in content, accompanied by extensive bibliographies to the pertinent literature.

This book constitutes a source material for a tremendous amount of information, otherwise difficult to obtain, in this broad field.

ALBERT SEGALOFF, M.D.

Manual of Clinical Laboratory Methods: By Opal E. Hepler, Ph. D., M. D., with a foreword by Dr. James P. Simonds. 4th ed. Springfield, Illinois, Charles C. Thomas, 1949. Illus. 387 p. Price $8.50.

This book is written primarily for technicians but there are important suggestions for physicians, notably the procedure for collection of sputum for examination. The chapters are divided into urinalysis, hematology, gastric analysis, liver function tests, bacteriology, mycology, serology, clinical chemistry, and procedures which the pathology technician is infrequently called upon to do such as preparation of allergy extract and electrocardiography. The tests are arranged in a form easy to follow. There is a discussion of the test, followed by detailed description of the procedure, calculation of results, and interpretation and sources of error. The plates on blood cells have been copied and appear to be slightly faded. Some statements might be questioned, i.e., the use of a 10 gauge needle for sternal marrow aspiration, marrow smears are more difficult to stain because of the fat; presence of Cabot rings after splenectomy; increased acid phosphatase in Paget's disease and acute leukemia. The chapter on blood groups including the Rh, is well presented and fairly well up to date. Some procedures should be brought up to date, such as the hypochlorite digestion of sputum for acid fast bacilli, the use of frogs in pregnancy diagnosis, blood chlorides and bromides. It is amazing that a book which has been used in numerous military hospitals, should have so little on venereal diseases with no mention of Donovan's bodies or the Frei test. The famous histochemist Gomori has been misspelled on several occasions. No references are given.

PHILIP PIZZOLATO, M. D.

Posttraumatic Epilepsy: By A. Earl Walker, M. D. Springfield, Illinois, Charles C. Thomas, 1949. Illus. 86 p. Price $2.75.

Essentially, this monograph is a brief account of Dr. Walker's surgical experience with cases of focal epilepsy among the wounded of the U. S. Army in the Second World War.

The discussion of etiologic factors and pathogenesis is followed by a review of the electroencephalographic and clinical aspects of the disease. The sections devoted to medical and surgical therapy are nicely done.

As a whole, this is an excellent book dealing with the problems of focal epilepsy ensuing from trauma in adulthood. It is particularly recommended to neurosurgeons and neurologists engaged in the management of posttraumatic epilepsy.

F. GARCIA BENGOCHEA, M. D.

Haemolytic Disease of the Newborn: By M. M. Pickles, M. D. Springfield, Illinois, Charles C. Thomas, 1949. Illus. 181 p. Price 15s.

This is a scholarly detailed study of the disease. Dr. Pickles goes into the history of the disease, the Rh factor, and its mechanism both in the mother, father, and baby. She then takes up the clinical forms of the disease and its treatment, and ends the discourse with the sequelae in the baby.

Dr. Pickles brings forth the many theories on this condition, she knows in what a confused state we still are in regard to the disease. She tries to give a clearcut picture of the treatment so as to help the clinician. She also brings out the fact that antibodies in mothers' milk can be destroyed by boiling and that they are rarely found in the milk after the second week of lactation. This will be a comfort to the mother who wishes to nurse her baby and to whom we have previously denied the opportunity. There is so much to be obtained from this book that it will take constant reference to digest it.

SUZANNE SCHAEFER, M. D.

Diseases of the Heart: By Charles K. Friedberg, M. D. Philadelphia, W. B. Saunders Co., 1949. Illus. 1081 p. Price $11.50.

The recent rapid advances in cardiology have necessitated a new orientation in the clinical approach to the diagnosis, prognosis, and treatment of diseases of the heart. Physiology and pathologic physiology have entered the arena of the everyday practice of cardiology via the several instruments of precision which have become workable tools in the hands of the clinician. This, in brief, is the raison d'etre of this big, well written, well edited and well gotten up book.

The plan of presentation is novel but thoroughly thought out and presented with lucidity and authority. The author is justified in his remark that "this book endeavors to provide a comprehensive and integrated exposition of the diseases of the heart." It is most important and interesting to see congenital heart disease presented from the point of view of curative therapy and bacterial endocarditis discussed as a curative disease. These

are but two of the near miracles that have been wrought in recent years and to which this book bears witness. If one should question the need for a reorientation, he need only compare this book with one of twenty-five years ago. There is no chapter devoted to the consideration of the normal heart and no general discussions as separate subjects of electrocardiography, cardiac catheterization, and angiocardiography. Instead they find their places in the discussion of the various diseases under discussion, which is probably as it should be. Or is it? Should a presentation of the normal heart have been alloted a separate chapter? I think so. Any clinician will admit that the normal and its variation with its wide range of anatomic and functional measurements is no mean problem.

One can discuss this book from many viewpoints. Authorities in every department of the subject may find something to which to take exception. Even the mode of subject arrangement may jar the notion of many. But this is the result of years of planning and thinking and comment should be held in reserve till one has read and pondered the volume. One thing then becomes certain. This book must have a wide appeal to men in all branches of medicine, clinical as well as investigative. Although this book begins with a consideration of circulatory failure, I feel sure that the author will not have to consider the failure of the circulation of his "Diseases of the Heart."

I. L. Robbins, M. D.

The Management of the Patient with Bronchial Asthma: By Maurice S. Segal, M. D. Springfield, Chas. C. Thomas, 1950. 158 p. Price $3.50.

This small monograph sums up very admirably the management of the patient with severe bronchial asthma. Physiologic principles are stressed throughout. An instructive feature of the book consists of a recapitulation of Segal's methodology for evaluating new drugs in asthma. Greatest stress is necessarily placed on pharmacologic agents in giving relief to the paroxysm. Allergic therapy is glossed over, though no fault could be found with the statement that emphasis on the allergic approach to management should not exclude physiologic therapy. A small inconsistency is noted for example, on page 21, for among the common offending allergens, house dust is not included. Yet on page 24 he states that "it has been our custom to routinely advise perennial nonspecific hyposensitization with house dust extract . . . for patients with negative skin tests . . ." He refers to patch tests for diagnosis of bronchial asthma; patch testing is exclusively beneficial in contact dermatitis. In the reviewers opinion better insight into the role of skin testing in allergic conditions might change for the better the author's attitude. But these are trivial points. The book can be recommended to all who would treat patients with severe asthma in the ways that are currently considered soundest.

Vincent Derbes, M. D.

Transactions of the American Goiter Association, 1949: Annual session May 26-28, Hotel Loraine, Madison Wisconsin. Springfield, Ill., Charles C. Thomas, 1950. Illus. 460 p. Price $10.50.

These are the papers delivered at the annual meeting of the American Goiter Association held in Madison, Wisconsin, May 26, 27, and 28, 1949. There has been a great change in the character of the subject matter presented at these meetings in the last decade. There are few surgical papers now; but many studies of physiology and pharmacology of the thyroid and many more on chemotherapy and nuclear therapy, innovations which are making rapid strides in this field. Radioiodine has found a place in the treatment of thyroid carcinoma metastases, and it makes a bid to replace surgery in certain cases of hyperthyroidism. Antithyroid drugs are of greatest value in preparation of patients for surgery. A symposium on thyroid carcinoma again points out the inherent potentials in this regard, in single adenomas of the of the thyroid gland.

Howard Mahorner, M. D.

Electrocardiography, Fundamentals and Clinical Application: By Louis Wolff, M. D. Philadelphia, W. B. Saunders Co., 1950. Charts, 186 p. Price $4.50.

This book adds another to the sizeable list of books designed for the beginner in electrocardiography. As expressed in the preface, the author has found that mere presentation of patterns is inferior to teaching basic principles. The book is divided into two sections, the first of which presents fundamental concepts, and the second relates these to clinical electrocardiography.

Section one is lucidly written and successfully presents the essentials of the theory of modern electrocardiography. Controversial issues, however, are not so labeled, and their detailed discussion is not possible or even desirable in a work of this type. Diagrams in this section are adequate.

Section two attempts to relate theory to practice, mainly by means of selected tracings with the author's interpretations and explanations. The tracings are well chosen to illustrate major points, but a considerable number are not effectively reproduced.

The term "arborization block" is used without a clear definition of the electrocardiographic features which the author considers indicative of this condition. The reviewer takes exception to the statements that ST segment and T wave changes are more reliable signs of infarction than are abnormalities of the QRS complex and that ab-

normal tracings may be encountered during illnesses which have nothing to do with the heart.

The omission of any discussion of arrhythmias, although on logical grounds, limits the usefulness of the book to the beginning student.

J. A. ABILDSKOV, M. D.

Current Therapy, 1950: Edited by Howard F. Conn, M. D. Philadelphia, W. B. Saunders Co., 1950. 735 p. Price $10.00.

This is the second in series of annual volumes, the purpose of which is to provide brief but complete outlines of currently accepted and practical treatment of almost any medical or surgical disease that a physician may encounter. The 250 contributors, including those from New Orleans—Drs. Vincent Derbes, Conrad G. Collins and Ernest C. Faust, are each outstanding in their particular fields. In diseases when treatment is still empiric or controversial, two or more contributors have been asked to describe plans of treatment.

There is no discussion of diagnosis or differential diagnosis and treatment is presented in clear, concise but complete, outline form. The material is divided into sections under body systems.

The volume is attractively bound; the type is legible in two columns on good paper. It is well indexed and lends itself to quick reference. It should be a valuable addition to the library of any practicing physician.

ALTON OCHSNER, JR., M. D.

The Salt-Free Diet Cook Book: By Emil G. Conason, M. D., and Ella Metz. Lear, 1949. 137 Pp. Price, $3.00.

By this time the indications for the diet are known to all clinicians. In this book the doctor's prescriptions are translated into the terms of the kitchen. The why and the wherefore of the diet are explained and the routine tables of sodium contents are present. Attention dietitians—home and hospital.

I. L. ROBBINS, M. D.

A Textbook of Surgery by American Authors: Edited by Frederick Christopher. Fifth ed. W. B. Saunders Co., Philadelphia & London, 1949. Illus., 1550 p. Price $13.00.

In this new edition of a textbook on surgery, which has long been an outstanding source of information for the student and practitioner, there are a large number of entirely new sections, and a number of new contributors. All in all, the chapters are excellent and are brought up to date. A few failed to meet the high standard set by the majority of the chapters, but this still remains an outstanding textbook of surgery, and it is highly recommended.

The reviewer fails to see the advantage of a new division of the pages into double columns. It is questionable whether this permits faster reading, and it looks more encyclopedic and less attractive. The illustrations remain excellent; many have been changed, among which are those accompanying the chapter on the breast. These, considering certain clinical features in the diagnosis, are, like other such changes, more instructive, particularly to students.

HOWARD MAHORNER, M. D.

PUBLICATIONS RECEIVED

The Commonwealth Fund, New York: Maternity Care in Two Counties, Gibson County, Tennessee, and Pike County, Mississippi, by Frank E. Whitacre, M. D., and Ellen Whiteman Jones, M. P. H.

The New York Academy of Sciences, New York: The Ground Substance of the Mesenchyme and Hyaluronidase, Volume 52, published by the Academy.

Oxford University Press, Inc., New York: British Medical Bulletin, Volume 5, published by Medical Department of The British Council; Clinical Electro Cardiography, by Francis F. Rosenbaum, M. D.; Management of Peripheral Arterial Diseases, by Saul S. Samuels, A. M., M. D.

W. B. Saunders Company, Philadelphia: A Textbook of Gynecology (6th Edition), by Arthur Hale Curtis, M. D., and John William Huffman, M. D.; Medical Diagnosis, Applied Physical Diagnosis (Second Edition), edited by Roscoe L. Pullen, M. D., F. A. C. P.

Charles C. Thomas, Publisher, Springfield, Ill.: Asphyxia Neonatorum, by William F. Windle, Ph.D., Sc.D.; Light Therapy, by Richard Kovacs, M. D.; Essay on The Cerebral Cortex, by Gerhardt von Bonin, M. D.